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THE
LARYNGOSCOPE.

VOL. LVII FEBRUARY, 1947. NO. 2

A REVIEW OF TUBERCULOSIS IN THE FIELD OF OTOLARYNGOLOGY FOR THE PAST YEAR.

FRANK R. SPENCER, M.D.,
Boulder, Colo.

Bueno[1] gave a brief review of the medical, surgical and physiotherapeutic approach to the problem of relieving pain in laryngopulmonary tuberculosis, with brief mention of the advantages and disadvantages of cocaine injection, alcohol injection with forcipressure, and electroanalgesic procedures.

Finally, Bueno refers to a new medication used parenterally which acts over the centers of pain perception or over the pathologic nerve endings, the pseudoneuromas of Dansac.

This is a solution called the compound A of Gillot or injection solution A (1 percent or 33 drops of compound A in 100 cc. of bidistilled water, previously filtered through wax). This solution of Gillot contains a purified oil or resin with an optimum pH. Experimental injections of rabbits caused no changes and practical applications to many tuberculous and nontuberculous patients were well tolerated. The advantage of the procedure is that it is not necessary to know the topography of the lesion, laryngoscopically. Patients were treated regardless of their condition; some with extensive lesions, and the general condition was not affected. The relief of pain was always successful; however, some phthisiologists say that at times it may provoke slight shock which can aggravate all the lesions and, therefore, should not be used

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on very sensitive patients because of perifocal reactions which can hasten death.

Le Lourd[2] believed his technique of novocain infiltration into the carotid sinus deserves a prominent place in treatment. It is as follows:

After locating the carotid pulse, at the level of the outer superior angle of the thyroid cartilage, a fine needle is introduced until it comes in contact with the artery. To assure adequate flooding of the region, 20 cc. of 1/200 novocaine solution are injected. Barring gross errors, there is no risk of injury to the large jugular vein or pneumogastric nerve, providing the needle is fine. By withdrawing a little, the injection can be increased, unless it is preferred to repeat the injection at another time.

Pain was almost immediately alleviated, often by anesthesia of a single sinus in bilateral affections; however, when a single infiltration proved insufficient, bilateral injections could easily be made and infiltrations could be repeated for months and remain efficacious.

Laryngeal examination after injection showed appreciable ischemia, predominantly on the infiltrated side, beginning at the periphery of the aryepiglottic folds within two to three minutes and slowly reaching the median line. The posterior pits were affected by the tenth minute. Secondary vasodilatation, which is important, was still considerable on the fifteenth day. The action of the drug lasted at least five days, generally seven to 10 days and rarely exceeded 20 days. Four or five injections were sometimes necessary, since the drug has a cumulative effect. The injection had no effect on the ulcerations and local processes.

This treatment was given to 19 patients for nine months. Nine had nonpainful laryngitis and the treatment was ineffective nine times. In 10 cases there were ulcerations accompanied by severe pain. One failure was recorded. It was a case of laryngeal resorption in which only three injections could be given before death.

A particularly brilliant result was described in the case of an asphyxiated patient admitted during a severe crisis of suffocation. She showed bilateral tuberculosis with large flocculations and multiple small ulcerations. Auscultation did not show the slightest physiologic pulmonary activity. The glottis was reduced to the caliber of a goose feather. The ventricular bands and the aryepiglottic folds were extremely edematous. The expected emergency tracheotomy was delayed by opiates. Three sinus injections on both the right and left sides were made within 15 days to unlock the larynx and re-establish a sufficient glottic orifice. Examination showed considerable reduction of the anterior edema, and for the first time in several months the patient could stretch out full length. Cyanosis disappeared at the same time that pulmonary symptomatology became enriched, following establishment of adequate arterial flow. This lasted nine months. Although a left collapse had been possible, the larynx actually remained atonic, ulcerated nonpainful and showing no recovery. Pulmonary lesions were extensive.

Auerbach[3] believed that much could be learned from the gross and microscopic laryngeal lesions at autopsy. He based his studies on 304 autopsies showing involvement of the larynx taken from 811 autopsies during a five and one-half year period. He found the larynx involved in 37.5 per cent of patients who had died of tuberculosis. He quoted the percentages of several other reports.

The ages ranged from 15 months to 82 years, with the majority in the age group from 20 to 39 years. There were 208 males and 96 females. Patients who had a rather rapid course for pulmonary tuberculosis, which was fatal in about two years, showed a higher percentage of laryngeal tuberculosis. In all but two of his cases the route of infection was direct. In 47 per cent of his patients with chronic pulmonary tuberculosis there was hematogenous tuberculous dissemination. Hematogenous infection of the larynx occurred in two of the cases. The basic lesion was an ulcer. In 19 of the 304 cases there were no gross lesions but only microscopic lesions. The ulcers involved almost all parts of the larynx and tra-

chea. He described in detail the superficial and deep ulcers and the histopathology of these and the other lesions. Ulcers were found most frequently on the true cords in 47.1 per cent of the cases.

Myerson[4] reported the use of sulfonamide spray for pain in tuberculous laryngitis with marked relief from pain, especially at meal time. He believed the pain was due to secondary infection more than to the tubercle bacillus. Pain from perichondritis was not relieved by the spray. Each application consisted of 5 to 10 gr. of the sulfonamide powder used by spraying with a powder atomizer while the tongue was held forward. He treated 60 patients in this way. Fourteen hopeless cases showed improvement and relief from pain. He reported two cases briefly, but he does not present these as cured.

Hulse[5] believed tuberculosis is characterized by its protean complications. He believed it is the most prevalent disease. Better treatment for pulmonary tuberculosis has lessened the laryngeal complication. He mentioned chemotherapeutic remedies, such as the sulfa drugs and their failure to be specific treatment. The author discussed menthol in oil, chaulmoogra oil, silver nitrate, lactic acid, ultraviolet rays, diet, Roentgen rays, galvanocautery, local and general rest, etc. He emphasized vocal rest as of more importance than any of the other methods of treatment. He reported briefly six cases to substantiate the value of vocal rest and placed this as only secondary to pulmonary control.

Humphries[6] emphasized the importance of laryngeal tuberculosis in any hospital or sanitorium for the care of the tuberculous. He found that collapse therapy has improved the treatment for pulmonary and laryngeal tuberculosis and it has lessened the incidence of the laryngeal complication. He referred to the two previous five-year surveys of results at the Virginia State Tuberculosis Sanitorium. He mentioned the importance of routine laryngeal examinations at frequent intervals for the tuberculous and the clinical lesions usually found. His five-year study from 1939 to 1944 of 139 cases of

laryngeal tuberculosis in 2,213 patients discharged from the sanitorium represented an incidence of 6.2 per cent. This was more than 50 per cent lower than in the two previous five-year studies.

There were 18 tables in Humphries' article and 15 references. He discussed galvanocauterization, laryngoscopy, biopsy, removal of the epiglottis, injection of the superior laryngeal nerve, tracheotomy, bronchoscopy and collapse therapy under surgical treatment.

Robin[7] treated patients with laryngeal tuberculosis showing ulcerative and granulomatous lesions by local applications of promin. A 20 per cent solution of promin in 50 per cent glycerine was sprayed into the larynx with a DeVilbiss spray. An application of two to four puffs was given once a day. Alternate cases were sprayed with chaulmoogra oil once daily. The spray with promin was continued for two weeks after the larynx showed marked improvement. Other methods of treatment such as silence, whispering, the galvanocautery, anesthetic powder, diet, vitamin C tablets, etc.

He gave 14 brief case reports of the patients treated with promin and of seven treated with chaulmoogra oil. In one patient the larynx was cured with promin in four weeks. The longest period of treatment was three months. The treatment was stopped after three months if patients didn't show improvement. Seven of the 14 patients treated with promin showed marked improvement. With chaulmoogra oil, five of the 12 patients were improved. The author doesn't believe that there is a great therapeutic advantage of promin over chaulmoogra oil, but that promin did give slightly better results. The author showed 28 colored illustrations of the larynx.

Figi, Hinshaw and Feldman[8] reported a case of laryngeal tuberculosis cured in approximately six weeks by using streptomycin as a laryngeal spray and by intramuscular and hypodermic injection. The patient was a married woman, 32 years old. She had relatively little pulmonary tuberculosis. The total amount of streptomycin administered parenterally was 34 gm. in 45 days. The dose administered parenterally varied

from 0.8 to 1 gm. (800,000 to 1,000,000 S units) of streptomycin hydrochloride per 24 hours divided into eight equal doses and given every three hours. For nebulization, 0.5 gm. of streptomycin hydrochloride was dissolved in 30 cc. of normal saline solution and 3 cc. of this administered at the beginning of each hour for 10 hours each day.

This method of treatment has given the best and the quickest result of any known method. The larynx has returned to normal and the subsequent follow-up shows that she has been cured.

At the meeting of the American Academy of Ophthalmology and Otolaryngology in Chicago, Oct. 13 to 17, 1946, Dr. Figi reported four more cases cured by streptomycin. This last paper will be published in the *Transactions* of the Academy. The writer had an opportunity to hear this paper read and he had the honor of discussing it. The treatment with streptomycin is one of the most outstanding contributions to the treatment of tuberculosis in many years. Unfortunately, if patients are given very much streptomycin, over a very long period, they may have vertigo and a hearing loss. The labyrinthitis may be toxic from impurities in the product. The product may and very likely will be improved to eliminate this objection.

REFERENCES.

1. Bueno, F.: Therapy for Relief of Pain in Pulmonary Laryngeal Tuberculosis. Realization of Effective Measures. Some New Treatments: Advantages and Disadvantages. *Rev. Assn. Med. Argent.*, 59:662-663, June 15, 1945.

2. Le Lourd: Carotid Sinus Infiltration in Laryngeal Tuberculosis. *Jour. de med. de Bordeaux*, 123:174-175, May, 1946.

3. Auerbach, Oscar: Laryngeal Tuberculosis. *Arch. Otolaryngol.*, 44: 191-201, Aug., 1946.

4. Myerson, Mervin C.: Tuberculous Laryngitis. *Jour. A. M. A.*, 130: 1014-1015, Apr. 13, 1946.

5. Hulse, Wm. F.: Treatment of Tuberculosis of the Larynx: An Evaluation. *Arch. Otolaryngol.*, 43:578-585, June, 1946.

6. Humphries, M. K., Jr.: Laryngeal Tuberculosis. *Dis. Chest*, 12:129-146, Mar.-Apr., 1946.

7. Robin, Ian G.: Treatment of Laryngeal Tuberculosis with Promin (Promanide). *Tubercle*, 26:186-189, Nov.-Dec., 1945.

8. Figi, Frederick A.; Hinshaw, H. Corwin, and Feldman, Wm. H.: Treatment of Tuberculosis of the Larynx with Streptomycin: Report of Case. *Proc. Staff Meet., Mayo Clin.*, 21:127-130, Mar. 20, 1946.

SYMPOSIUM ON NOISE.

(a) THE PERCENTAGE OF CAPACITY TO HEAR SPEECH, AND RELATED DISABILITIES.*

EDMUND PRINCE FOWLER, M.D.,
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Five years ago before this Society I described a method for measuring the binaural percentage loss of capacity to hear speech, and suggested standards for use in the Army, Navy and Air Corps. In this and subsequent papers the philosophical and factual backgrounds of the procedure were set forth in detail. I will not repeat them at this time.

In brief the method consisted in testing both ears by standard audiometric methods and weighting the frequencies according to their relative importance for hearing and understanding speech, in this way obtaining a weighted decibel loss for each ear. This weighted decibel loss was used in connection with a table (see Fig. 1). The table was constructed so that if a vertical line was drawn from the figure indicating the weighted decibel loss for the better ear, and a horizontal line was drawn from the figure indicating the weighted decibel loss in the poorer ear, the point of intersection of these lines would indicate the overall percentage loss in capacity.

The reason for using the table was to enable one to allow not only for the changing increments of loss with different degrees of deafness in both ears, but also the changing ratio of importance of the hearing in the better and the poorer ears with differences in binaural threshold levels. This method took into consideration the recruitment phenomenon. The figures printed at the right side of the table indicate the subtractions and additions that should be made for this factor. Dr. James Hardy Neil has adopted the method for use in the New Zealand Army, and to conserve time has prepared

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charts showing complete percentage figures at all degrees of loss.

From the standpoints of factuality and accuracy, I still see no reason for making any change in the method. I stressed this forcibly before the Committee on Audiometers and Hearing Aids of the American Medical Association, but my method

Fowler's Table for Estimating Percentage Loss of Capacity for Hearing Speech in Monaural and Binaural Deafness

Weighted Decibel Loss—Worse Ear	Weighted Decibel Loss—Better Ear										
	0	10	20	30	40	50	60	70	80	90	100
0	0%										
	0										
10	0	2									
	1	5									
20	1	3	9								
			14								
30	2	4	12	20							
				27							
40	3	5	14	24	35						
					44						
50	4	6	15	27	40	54					
						63					
60	5	7	16	29	44	59	71				
							78				
70	6	8	17	30	47	63	76	84			
									89		
80	7	9	18	31	49	66	80	88	93		
										96	
90	8	10	19	32	50	68	83	91	96	98	
											99
100	9%	11	20	33	51	69	85	93	97	99	100%

Percentage Loss of Capacity

Recruitment Factor

Apply corrections before averaging A.C. losses

Subtract from A.C. loss, for B.C. loss	
<15 db.,	0
15	5
20	4
25	3
30	2
35	1
40	0
Add to A.C. loss, for B. C. loss = 45	2
50	5
Over 50	5

Weighted decibel loss is the sum of the corrected A.C. losses at the following frequencies, times the corresponding percentage factors:

512	1,024	2,048	4,096
15%	30%	40%	15%

The percentage loss will be indicated at the intersection of a vertical line from decibel loss in better ear with a horizontal line from decibel loss in worse ear

(Test in a soundproof room)

A.C. indicates air conduction, and B.C., bone conduction.

Fig. 1.

was not adopted as a standard, because it seemed to the majority of the committee to be too complicated. Well, there are some things that are intrinsically so complicated that you cannot figure them in a very simple manner, and testing the capacity to hear speech is one of these. The only calculation in my method, however, which really may be called complicated or laborious appears to be the multiplication of the decibel losses at each of the frequencies by the weighting figures. This may be overcome in three ways: *1.* By multiplying all possible decibel losses by the weighting figure and putting the products in a table by itself (see Table 1), thus avoiding the

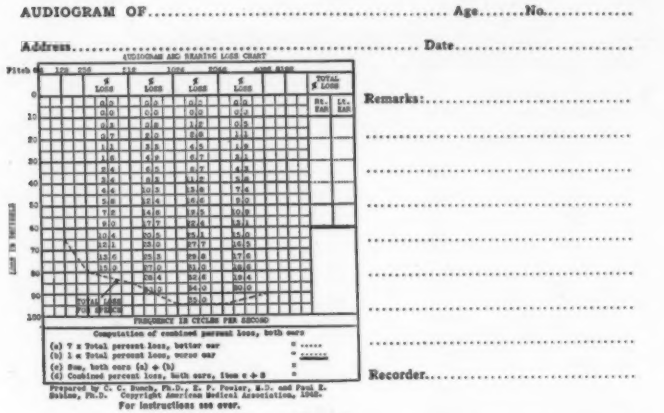
necessity of actual multiplication; *2.* by printing these figures (the weighted decibel losses) on the audiogram chart, over or just above the threshold plotting at each frequency. As a matter of fact, this all seems rather unnecessary because to multiply by 15 is no more difficult than to multiply by 12 after a little practice. To multiply by 30 it is really only necessary to multiply by the 3 and suffix the 0 (and in like manner with 40); *3.* by changing the weighting of the frequencies, making the weighting of 512 = 20 per cent instead of 15 per cent, and 4.096 = 10 per cent instead of 15 per cent, thus avoiding difficulty in multiplication in every instance.

TABLE 1. SHOWING THE PRODUCT OBTAINED BY MULTIPLYING THE VARIOUS DECIBEL LOSSES BY THE WEIGHTING FIGURES.

Speech Frequencies Weightings Db. Losses	512 15%	1,024 30%	2,048 40%	4,096 15%
10	1.50	3.00	4.00	1.50
15	2.25	4.50	6.00	2.25
20	3.00	6.00	8.00	3.00
25	3.75	7.50	10.00	3.75
30	4.50	9.00	12.00	4.50
35	5.25	10.50	14.00	5.25
40	6.00	12.00	16.00	6.00
45	6.75	13.50	18.00	6.75
50	7.50	15.00	20.00	7.50
55	8.25	16.50	22.00	8.25
60	9.00	18.00	24.00	9.00
65	9.75	19.50	26.00	9.75
70	10.50	21.00	28.00	10.50
75	11.25	22.50	30.00	11.25
80	12.00	24.00	32.00	12.00
85	12.75	25.50	34.00	12.75
90	13.50	27.00	36.00	13.50
95	14.25	28.50	38.00	14.25
100	15.	30.	40.	15.

Instead of adopting my method, the special committee, then consisting of Dr. Bunch, Dr. Sabine and myself, recommended a method which printed the weightings in what purported to be the average of the per cent loss between each of the contiguous decibel losses at each of five frequencies (256, 512, 1,024, 2,046, 4,098) (see Fig. 2). The average was determined by drawing a straight line between the adjacent octave losses in each case, and estimating the average loss from the percentage loss corresponding to the midpoint of this line.

The per cent losses at these four midfrequency points were added for each ear. This then represented the per cent loss for each ear. To obtain the binaural per cent loss, the total per cent loss in the better ear was multiplied by 7, the total per cent loss in the poorer ear was multiplied by 1, and the addition of the two multiples was divided by 8, thus arriving


AUDIOGRAM OF..... Age.....No.....

Address..... Date.....

AUDIOGRAM AND HEARING LOSS CHART

Pitch cps 128 256 512 1024 2048 4096 and over

% LOSS | % LOSS | % LOSS | % LOSS | TOTAL % LOSS

Rt. Ear | Lt. Ear

LOSS IN DECIBELS

0 10 20 30 40 50 60 70 80 90 100

Total % Loss

FREQUENCY IN CYCLES PER SECOND

Computation of combined percent loss, both ears

(a) 7 x Total percent loss, better ear = ...

(b) 1 x Total percent loss, worse ear = ...

(c) Sum, both ears (a) + (b) =

(d) Combined percent loss, both ears, Item c ÷ 8 =

Prepared by C. C. Bunch, Ph.D., E. P. Fowler, M.D. and Paul P. Sabine, Ph.D. Copyright American Medical Association, 1942.

For instructions see over.

Remarks:.....

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Recorder.....

Fig. 2.

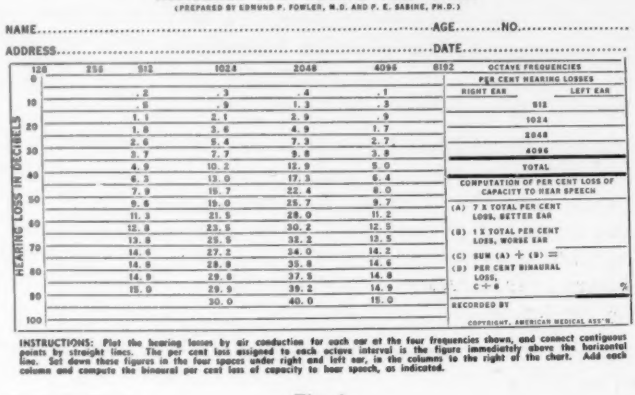
Instructions: Plot measured hearing losses for each ear at different frequencies. Connect successive points by straight ruled lines. Percentage loss assigned to each octave interval is figured in space immediately above straight line in that octave. (When two or more spaces are intersected take average of figures in intersected spaces.) Set down figures for four octaves for each ear in columns to right of chart. Compute for combined percentage loss, both ears, as indicated.

at a binaural overall percentage loss of hearing by this method.

Clinical experience showed that the results were far from accurate, being at least 20 per cent too low in certain types and degrees of deafness. To correct these underestimates, I recommended that the percentage figures should be changed so that when they were added they would correspond within 1 or 2 per cent with the percentage losses derived from equal weighted decibel losses in my table, and that the figures should be placed on the ordinates of four frequencies (512, 1,024, 2,048, 4,098), thus avoiding the necessity of averaging

the losses at contiguous octave frequencies. This enabled us to retain the American Medical Association chart method, but it did not provide a true allowance for the effects from differences in the hearing of the two ears.

A year and a half ago this arrangement was approved by the committee then consisting of Dr. Hughson, Dr. Sabine and myself, and later by the Consultants on Audiometers and Hearing Aids of the American Medical Association; however,



AUDIOGRAM AND HEARING LOSS CHART

(PREPARED BY EDMUND P. FOWLER, M.D. AND P. E. SABINE, PH.D.)

NAME..... AGE..... NO.....

ADDRESS..... DATE.....

HEARING LOSS IN DECIBELS	128	256	512	1024	2048	4096	8192
0							
10			.2	.3	.4	.1	
20			.5	.9	1.3	.3	
30			1.1	2.1	2.9	.9	
40			1.8	3.6	4.9	1.7	
50			2.6	5.4	7.3	2.7	
60			3.7	7.7	9.8	3.8	
70			4.9	10.2	12.9	5.0	
80			6.3	13.0	17.3	6.4	
90			7.9	15.7	22.4	8.0	
100			9.6	19.0	25.7	9.7	
			11.3	21.5	28.0	11.2	
			12.8	23.5	30.2	12.5	
			13.8	25.5	32.2	13.5	
			14.6	27.2	34.0	14.2	
			14.8	28.8	35.8	14.6	
			14.9	29.6	37.5	14.8	
			15.0	29.9	39.2	14.9	
				30.0	40.0	15.0	

OCTAVE FREQUENCIES PER CENT HEARING LOSSES	RIGHT EAR	LEFT EAR
512		
1024		
2048		
4096		
TOTAL		

COMPUTATION OF PER CENT LOSS OF CAPACITY TO HEAR SPEECH

(A) 7 X TOTAL PER CENT LOSS, BETTER EAR

(B) 1 X TOTAL PER CENT LOSS, WORSE EAR

(C) SUM (A) ÷ (8) =

(D) PER CENT BINAURAL LOSS, C ÷ 8 %

RECORDED BY

INSTRUCTIONS: Plot the hearing losses by air conduction for each ear at the four frequencies shown, and connect contiguous points by straight lines. The per cent loss assigned to each octave interval is the figure immediately above the horizontal line. Set down these figures in the four spaces under right and left ear, in the columns to the right of the chart. Add each column and compute the binaural per cent loss of capacity to hear speech, as indicated.

Fig. 3.

the majority in both instances clung to the unchangeable ratio of 7:1 between the better and the worse ears (see Fig. 3).

In pure conduction lesions the revamped ratings will produce results corresponding closely with those obtained by the use of my table for equal binaural deafness, because they were in fact taken from the table at the line of the equal binaural loss, but making 1 or 2 per cent changes at some levels of loss to enhance the accuracy of the method.

The reason that the American Medical Association method is not satisfactory is in part due to the use of the unchangeable 7:1 ratio for multiplying the per cent losses in the better and poorer ears.

A ratio of 7:1, or any other fixed ratio, is in my opinion illogical. It does not represent the relative importance of the better and worse ears at the different levels of hearing, because the relative importance changes not only with the degrees of deafness in each ear, but with the differences between the degrees of deafness in each ear.

Any unchangeable ratio ignores the fact that the worse ear becomes relatively less and less important for hearing as its hearing goes down, whereas the better ear becomes relatively more and more important as its hearing goes down. Using a fixed ratio ignores these changes. To allow for them, we must use some method to obtain a changing ratio. To overcome the difficulty and still employ the committee's chart, I recommend the following procedure:

Use the figures 10 and 1 to represent the importance of the better and the worse ears, respectively, if the better ear does not show more than a 10 per cent hearing loss, and for losses in the better ear of more than 10 per cent to vary this ratio by making the figure used to multiply the percentage loss in the worse ear correspond with the figure representing one-tenth the loss in the better ear.

The directions beneath the audiogram chart would then read as follows:

DIRECTIONS FOR DETERMINING BINAURAL LOSS IN CAPACITY TO HEAR SPEECH.

A) Multiply the % loss in better ear by 10. = .

B) Multiply the % loss in poorer ear by (1/10th loss better ear) . = .
(If loss in better ear is less than 10% multiply by 1.)

C) Add A and B and divide by sum of the multipliers ..... .).

D) The binaural % loss..... . % loss

This method will alter the calculations so that the results will correspond within 1 or 2 per cent to the figures shown on my table for equal binaural losses in hearing.

Let us compare the results in an unequal binaural loss of hearing, using the latest A.M.A. chart with its unchangeable 7:1 ratio, and then the same chart using my variable ratio.

If there is a loss in one ear of 54 per cent, and in the other of 80 per cent, then:

Using ratio of 7:1		Using ratio of 10:5.4 (which is 1/10th loss better ear)	
Better ear loss 54%	× 7 = 378	54% × 10.	= 540
Poorer ear loss 80%	× 1 = 80	80% × 5.4	= 432.0
	8)458	15.4	)972.0
Binaural loss	57.2%		63.1%

It will be seen from the above that by multiplying the loss in the poorer ear by 5.4 (which is 1/10th the per cent loss in the better ear) we are able to increase the sum of the products from 458 as in the A.M.A. method to 972 (an increase of 514), and we are able to use a divisor of 15.4 which increases the sum of the multipliers 7.4, thus accomplishing our purpose to raise the addition of the products relatively more than the figure we use as a divisor.

If instead of using the A.M.A. method, we use my table and a weighted decibel loss of 50, which corresponds to a 54 per cent binaural loss, the per cent binaural loss would be 66 per cent. This is 8.8 per cent greater than the latest A.M.A. 7:1 method produces, and 2.9 per cent greater than the latest A.M.A. chart using the suggested variable multiples; however, if the hearing losses were due to nerve deafness, allowances for the recruitment blurring factor require that 5 db. should be added to the weighted decibel loss at each frequency, since this is indicated when the threshold loss is 50 db. or more.

In the example given, if all frequencies are down 50 db. from nerve deafness in the better ear the weighted decibel loss in the better ear would be 55 (50 + 5), and the weighted decibel loss in the poorer ear would be 65 (60 + 5). Applying these to my table, we obtain 68 per cent as the binaural per cent loss. It will be seen that this is 10.5 per cent over the estimate obtained from the latest A.M.A. method, and 4.9 per cent over that obtained by my changeable multiple method. If the 1942 A.M.A. directions were used the difference between the A.M.A. and my table would be about 20 per cent.

It would seem that clinically the figures obtained by either of my methods are nearer the true loss than those obtained by the A.M.A. method.

When a person has a loss in one ear of 80 per cent and a loss in the other of 50 per cent, the poorer ear is of little use in hearing ordinary conversation, and the better ear is also of little use if it is turned away from the source of sound. In lesser degrees of deafness, sounds reflected back from walls or furniture are often useful, and especially to the better ear.

At first thought it might seem that since the better ear (as long as it is the better ear) becomes relatively more and more important as the hearing in both ears goes down that the figure used to multiply its per cent of loss should be increased accordingly. In other words, that the percentage of hearing loss in the better ear should be multiplied by a larger and larger figure as its hearing goes down. This is not basically sound, and the solution is not as simple as it seems.

Let us take a concrete example. Say, the hearing in the better ear shows no loss, and that the hearing in the poorer ear is down 50 per cent, and does not change as the hearing in the better ear goes down. Then it is evident that when the better ear reaches a loss of 50 per cent, its hearing loss will coincide with the loss in the poorer ear (50 per cent). In other words, both ears are then equal in hearing and equal in importance. It is thus shown that the poorer ear becomes more and more important the nearer the approximation of the threshold of the two ears; it becomes more and more a factor in the calculations the nearer the percentages of hearing loss in the two ears coincide. (This is equivalent to saying that under these conditions the better ear becomes relatively less important).

It is evident that if both ears are down 70 per cent, or any other equal per cent, that the per cent of loss is equal in each, and that the ratios of importance are equal. This does not necessarily mean that the losses in each must be multiplied by the same figure. It is also evident that when the losses in the two ears are identical it is immaterial what ratios you

use as long as you divide the product of the percentage losses in the two ears by the sum of the figures used as multipliers. In all instances where the losses in the two ears exactly coincide, the two methods I have just outlined take care of this.

Consider another example; suppose the poorer ear is 20 per cent below the better ear at all frequencies, and that this difference between the two ears is maintained as the hearing in both goes down. At the start when the better ear is hearing normally there is no more than 1 or 2 per cent binaural loss in overall capacity to hear speech as derived from the A.M.A. chart, but when the better ear is down to 50 per cent, the poorer ear then being 70 per cent, the ratio has changed. From the initial 10:1 it has changed to 10:5 — in other words, it is now 2:1. And so it would seem that when the difference in hearing in the two ears remains the same at every level of increasing loss the multiplier figures (the ratio of importance figures) approach closer and closer. If the hearing in the originally better ear becomes worse than that in the originally poorer ear, the ears would change their relative importance accordingly.

These simple observations clearly show that if our calculations are to correspond to clinical experience, changing multiples are imperative. I have shown two ways to arrive at a logical answer: *1.* using a table such as mine, *2.* using a variable ratio 10:1 to 10:10, depending upon the per cent loss in the better ear. I know of no other simple ways to overcome the difficulties.

The table automatically changes the ratio, without using any ratio figures. It takes care of this factor automatically, and also the factor of diminishing importance of the poorer ear with increasing binaural differences in hearing levels; however, even the changing 10:1 to 10:10 multiples do not take care of this latter factor because at every different level of loss in the better ear but one figure is used as a multiplier of its loss, and but one figure is used as a multiplier of the loss in the poorer ear, no matter what the hearing in the poorer ear or the differences between the two ears. I have been unable to overcome this deficiency under the limitations of the A.M.A. chart.

It is misleading to use the term "ratio of importance" because it is really not a ratio of importance we are using to multiply the losses, but only a multiplier which will produce the desired overall result under the conditions set up by the chart.

After obtaining a percentage of loss of capacity figure, how are we to employ it in determining the disability suffered? Frankly I do not know, and yet the percentage of capacity to hear the speech frequencies should be a major factor in determining disability.

The main difficulty is that no two people react the same to the same stimuli or the same handicap, or to its suddenness or rate of progression. In many instances a person with a major loss will suffer less disability than one with the minor loss. Some simply give up and throw themselves on the family or the community for support. Others persist in their job and make good, and in many instances attain great eminence in their fields of endeavor, no matter how severe their deafness, or other handicap.

In determining the handicap, the prognosis, and hearing and operative aids, come into the picture. A man is less handicapped if the prognosis is favorable, and if he can be helped by these means, than if he cannot be helped.

Prognosis is a matter of diagnosis and result of treatment. Operations are limited to favorable cases, such as those with uncomplicated otosclerosis.

In obstructive deafness even when complicated by severe neural lesions hearing aids are useful, but not in total or near total deafness. The greater the nerve deafness the less efficient the hearing aid in lessening the disability. Proper threshold audiograms with recruitment measurements will indicate whether A.C. or B.C. will be the more acceptable. If the B.C. is down less than 30 db., either an A.C. or B.C. aid may be chosen, the latter not because it is more efficient but usually because it is less conspicuous and leaves the meatus open. In such instances little amplification is needed for near conversation, but if the two most important speech frequency

regions (1,024 and 2,048) are depressed below 50 db. the unaided voice or the amplified voice must reach the ear with an intensity of 70-odd db. over normal threshold for speech to be heard with much satisfaction.

In nerve deafness the recruitment phenomenon is largely accountable for the fact that in most instances a flat response hearing aid is acceptable. The recruitment of loudness brings up the volume of the high tones, which are the ones usually most affected by the neural lesions, and if there is little or no nerve deafness for the low tones, and an impedance lesion, this latter automatically brings down the low tones without the necessity of suppressing them in the response of the hearing aid. If there is a nerve deafness for all of the speech frequencies, the recruitment phenomenon tends to equalize the responses at the different frequencies and so here also a flat response is preferred.

Except in a case where there is no deafness in the low tones little or no necessity exists for depressing the low tone response of the hearing aid. If it were not so, there would be more to the high, low and middle tone adjustments of aids than is now said to be necessary. In most instances the small adjustments in the so-called "fitting" of hearing aids have more sales value than hearing value; however, there are exceptions. We must remember to temper our judgment somewhat, depending on whether we are determining ability to take on a new job or ability to hold a job in which one has been and still is efficient. In other words, whether one is being hired or likely to be fired.

Taking all these things into consideration, it seems apparent that degrees of disability must be determined in the courts, using the percentage of hearing capacity as a basis for calculation, but only as a basis. The ability to overcome a handicap is often of greater importance than the handicap itself. Each case must be considered individually because the problem is never exactly the same in any two persons. If there is any doubt in the matter, it may be, as in baseball, we should favor the runner.

140 East 54th Street.

SYMPOSIUM ON NOISE.

(b) NOISE IN INDUSTRY.*

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The condition which we designate as "occupational deafness" has been recognized for many years. The early work on this condition was done on boilermakers. Hence the term "boilermaker's deafness." In 1890, Barr[1] reported that in making a survey of 100 boilermakers who had followed their occupation for more than three or four years, he did not find a single instance of normal hearing. During the period of the first World War, owing to the increased industrial production incident thereto, interest in this condition increased. In the field of industry it was recognized that machinists, locomotive engineers, tractor drivers, riveters and steel workers in general were suffering from a more or less severe form of deafness. With the tremendous expansion of the heavy industries during the recent war and particularly because of the accentuated use of such tools as the pneumatic hammer, the chipper and the riveting gun, especially when used in confined, reverberating areas, the importance of this form of deafness has assumed major proportions.

The effect of pistols, rifles and other sporting firearms in producing a similar form of deafness was known before the war. Of the large number of men subjected during the recent World War II to the noise of rifles, repeating rifles, machine guns and guns of larger caliber, irrespective of the blast effect from larger guns, mines and bombs, many will return to civilian life suffering from the same sort of deafness as that caused by the tools of heavy industry. The noise of the engines in planes and submarines has also exaggerated this problem.

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THE EFFECT OF NOISE ON THE EAR.

Experimental work on animals[2,3,4,5] teaches us that the organ of Corti is susceptible to loud noise and more particularly noise of high pitch and that it is more susceptible in the basal coil of the cochlea where high tones are localized. Lurie[6] calls attention to the fact that "the degeneration that takes place in these experimental animals is of the same type that occurs in animals which inherit deafness." As it is well known that in all cases of perceptive deafness, whatever the cause, the organ of Corti is more susceptible to degenerative processes in the region of the basal coil of the cochlea, it should not be surprising that the injurious effect of noise is also selective for this area. McCoy[7] calls attention to three factors which may operate to localize the greatest damage in the basal coil. First is the action of the intratympanic muscles, the tensor tympani and the stapedius, which "serve to protect the inner ear from damage from excessive amplitude of vibration which might be caused by intense tones of low pitch." (The quotation is from Stevens and Davis.[8]) The second factor is the greater exposure of nerve endings at the cochlear base where sound waves enter. The third factor is that there is a vascular bifurcation in this area, which, associated with a narrowing of the cochlear duct at this point is considered to be a predisposing weakness by Larsen.[9]

THE CHARACTER AND SOURCES OF NOISE.

While we must agree that noise of high pitch and of sufficient intensity when long continued is injurious to the hearing, we must not be stampeded into the belief that every loss of hearing for high tones in a factory worker is due to the noise or to the noise alone. We know that the causes of a perceptive type of deafness in the adult are manifold; furthermore, we must realize that all ears are not equally susceptible to the effects of noise. We must also realize that the adult is probably more susceptible to the deleterious effects of noise in direct proportion to his age. We must also consider the probability that the effect of noise may be more injurious to one with some pre-existing hearing defect than in one with

normal hearing. It is, therefore, very difficult to determine the importance of noise alone in the causation of any particular hearing defect.

We have no sure criterion of the intensity of noise which will produce a hearing defect. Bunch[10] thought that noise of from 80 to 90 db. was hazardous. This may be true if the noise is continued long enough, but Hallowell Davis[11] says that we have no rigid proof of permanent impairment of hearing by noises of less than 115 to 120 db. We can, I think, safely disregard the effect of noise below 100 db. of intensity; however, in connection with this noise of lesser intensity, we must give due consideration to hours, months and years' exposure and to the health and age of the worker. We know that the cochlear mechanism can suffer from fatigue engendered by noise and completely recover if a sufficiently long rest period is given, but we do not as yet know enough about the length of exposure from which recovery of the hearing function can take place.

Of the various sources of noise in industry, the chipping hammer leads the list. One hammer produces from 110 to 135 db. of noise, depending upon the material being worked and the power of the hammer (McCoy[7]). The noise level behind the hammer is somewhat less than it is near the hammer. The noise level is also greater in a confined space with sound reflecting walls than it is in a free field. The riveter is also exposed to a noise of 115 to 128 db. (McCoy[7]) and the noise level is greater on the other side of the plate where the rivet bucker is working with a small pneumatic hammer. The amount of noise produced by these operations varies with the heat of the rivet, being appreciably less when the rivet is hot and soft than when it cools off. The pneumatic hammer and the pneumatic drill are also sources of excessive noise. The intensity of the noise produced by all these operations rises to some extent with the number of operations going on at one time. McCoy[7] is author of the statement that when two operations are running at one time one may expect an additive effect of approximately 3 to 4 db. over that of one alone.

While we must admit that noise of excessive intensity and long continued can cause a hearing defect of the perceptive type, we have in the past had no rigid criteria that it will produce a hearing loss in every ear, nor have we had any definite proof that it was the sole cause in a hearing impairment. Numbers of workers in any given industry have been examined audiometrically and composite graphs of those examined have been plotted showing a certain degree of perceptive deafness. This has been done by Bunch,[10], Larsen,[9] McCoy,[7], Gardner[12] and others, but in most of these surveys there is nothing to show that the ears and the hearing of the workers had been accurately examined prior to their entry into the specific occupation. While indicative of the trend, these surveys, therefore, cannot be accepted as scientific evidence of the effect of noise upon the hearing.

Much more conclusive from a scientific point is a recent survey made by McCoy.[7] One hundred young individuals who were about to enter a noise occupation were audiometrically examined by McCoy. These men were mainly negroes from rural areas whose history and examination revealed no evidence of ear, nose and throat disease. After working for seven hours in chipping school where they were exposed to a noise intensity of 110 to 130 db., a representative group of the men had a distinct loss of hearing associated with tinnitus and a feeling of discomfort as indicated in the following chart (see Fig. 1). On the following day the hearing loss was only discernible by audiometric examinations and there was a relief from the associated symptoms. This initial result was probably the effect of fatigue and the spasticity of the intratympanic muscles and the recovery to the adaptability of the nervous system; however, after a month's exposure to this noise, there was a definite and permanent deficiency in the high frequency range as indicated in the next audiogram (see Fig. 2). This loss was not materially affected by a rest of one or two days.

To what extent a person may be exposed to noise and suffer only the hearing loss which goes with nervous fatigue, without actual destruction of hearing, has not yet been determined and

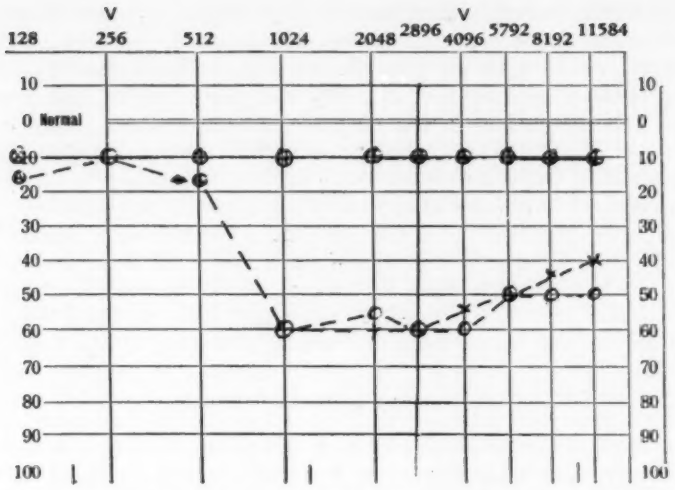

Fig. 1. Broken lines show levels after working seven hours in a chipping school. Taken from the paper of David A. McCoy.

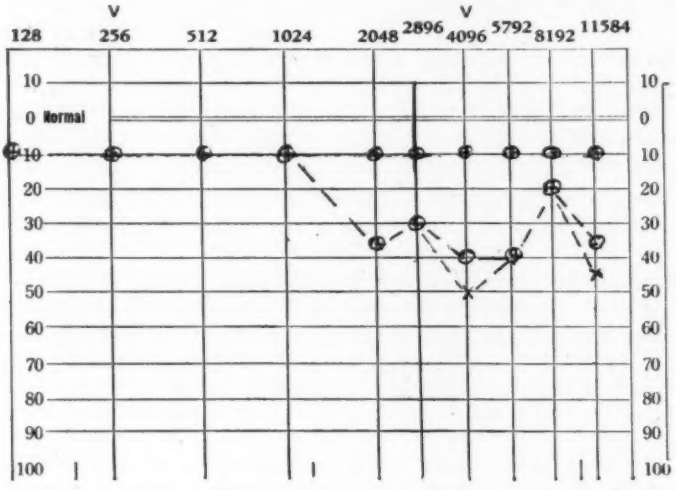

Fig. 2. Broken lines show the levels recorded after the man had worked one month in chipping school. Taken from the paper of David A. McCoy.

probably varies from individual to individual. Perlman[13] lists the following factors as important in this process: *1.* total time of exposure; *2.* length of exposure per period; *3.* loudness of sound stimulus; *4.* character of sound stimulus, continuous or interrupted; *5.* frequency of the noise; *6.* type of space, environment; *7.* protective devices used; *8.* age of the worker; *9.* previous aural disease; and *10.* constitutional factors such as differences in susceptibility.

NOISE REDUCTION AND EAR PROTECTION.

The noise of industry cannot be eliminated, but much can be done to reduce it and protect the ear against its effect. This can be accomplished in three ways: The first principle is the reduction of noise at its source. Just to enumerate a few of the things which can be done: Redesigning of machinery to reduce the vibration of unbalanced rotating or reciprocating parts; reduction of too liberal bearing tolerance which permits clanking rods and pounding shafts; change in materials such as the substitution of nonmetallic for metallic gears; change of machine layout so as to prevent the segregation of noisy machinery and to substitute noisy operations between quieter ones; removal of noisy machines from sound carrying structural beams; the reduction of impact noise on metal surfaces by the substitution of fibre, wood, linoleum, rubber or other nonmetallic substances; the use of cushion wheels on hand or power trucks; the substitution of asbestos-type or fibre panel for vibrating metallic sheets that produce annoying rattles; proper lubrication of moving parts; the tightening of all loose parts and the replacing of all worn parts; the education of shop personnel on the "work quietly" idea; the suspension of a machine on resilient mountings by using the elastic properties of such materials as cork, felt, rubber and asbestos; the prevention of the transmission of vibration by the use of the "floating floor" principle. This consists of the mounting on steel springs or other resilient material of entire floor sections so that they have absolutely no physical contact with the rest of the building except through the insulation.

The second principle involved in noise abatement concerns

the isolation enclosure of the noise source and the prevention of the spread of noise by absorption treatment of surrounding walls and ceilings. Especially noisy machine operations can be isolated from the rest of a building by surrounding them by sound absorbing walls and ceilings similar to the test rooms of airplane motors. The noise transmission loss of a wall or partition may vary from 25 per cent (poor) to 50 per cent (good), depending upon the materials used and the method of construction. Transmission loss is what engineers call a reduction in the decibel intensity of any sound passing through a wall or partition. A good isolation wall should reduce the transmitted noise to the outside noise level which may be around 70 to 80 db. In constructing an isolation enclosure the possibility of transmission of noise through floor or ceiling must not be overlooked. A concrete or tile floor which may have an excellent transmission loss for airborne noise may be an excellent transmitter of impact and vibration. These are problems which concern the acoustical engineer and not the otologist.

Noise reduction by the isolation enclosure principle is not limited to the complete separation of one or more noisy machines or processes. Hardly a department exists where the overall noise level cannot be reduced by merely suitably enclosing noisy parts of machines such as transmission parts, gears and other noise-producing mechanisms. Such special enclosures can also serve as safety guards.

Having more or less successfully treated the noise at its source, the acoustical engineer must concern himself with the problem of noise reflection from walls and ceilings. Hard and high density surfaces reflect sound or noise. Porous and low density surfaces tend to absorb noise instead of reflecting it and act as a noise sponge corresponding to their absorption coefficients. Acoustical facing materials are of four general types: *1.* plasters, *2.* blankets, *3.* boards and *4.* tiles, which are made from such nonabsorptive materials as mineral wool, hairfelt, glass wool, wood excelsior, rock wool, cork, asbestos and various vegetable fibres held together by a suitable binder. Such acoustical treatment of walls and ceilings re-

duced the overall noise 52 per cent in a factory room filled with punch presses, and 49 per cent in a large printing establishment.[14]

We come now to the third principle in a noise control program. Noise, to some extent, may be controlled or reduced at its source under the two foregoing principles, but in spite of that there are some manufacturing processes in which the noise cannot be reduced to a safe level and, as Sabine[15] says, "In spite of all that can be done, however, it must be admitted that factories where steel mightily strikes steel are bound to be noisy places." In situations where the noise at its source cannot be reduced to a safe level we must advise measures which will prevent it from reaching the ear at a dangerous level. This can be accomplished by the use of ear defenders or wardens, a subject which will be taken up by Dr. H. Marshall Taylor. "Numerous types have been devised in recent years. They range from treated cotton and wax plugs to plastic and rubber molds which fit the ear canals."[12] (Quoting from Taylor[16]): "A good ear defender must meet many requirements: It must be easy to insert and have good retention; it must be comparatively light in weight and unbreakable; it must decrease noise of high frequency without impairing to an important degree the hearing of commands. Cleanliness must especially be considered." The Navy has long advised gunners to place cotton in their ears when firing guns. Impregnating the cotton with oil or wax increases its efficiency. Other ear defenders are made of solid soft rubber and sponge rubber. The sponge rubber defenders work on the principle of absorbing the noise rather than blocking it out. Taylor speaks of a plastic defender developed at the Naval Gunnery School at Jacksonville, Fla., by Lieut. Cox and Lieut. Geller. This defender is very excellent in reducing noise but has the disadvantage that it must be individually molded.

I have recently been in communication with Hallowell Davis[17] concerning the V-51R Ear Warden which is now being manufactured for civilian use by the Mine Safety Appliances Company, of Pittsburgh, Pa. This ear warden,

according to Davis,[11] will attenuate noises by 30 db. or more and bring down the extreme noises to 100 db. or less.

It is said that workers rebel at wearing earplugs or defenders. The deterioration of hearing from the effect of excessive noises is a slow, insidious process. The process is slow enough that the worker does not realize that he is losing his hearing until irrevocable damage has occurred; moreover, the worker who is already hard of hearing cannot see why his residual hearing needs any further protection. This entire problem, it seems to me, is one of propaganda and education on the part of the medical profession and of management.

MEDICOLEGAL ASPECTS.

Occupational disease and trauma, quite apart from isolated accidents, with its resulting loss of function, is now compensable in most of the states. We recognize as a fact that under certain conditions the hearing function can be damaged by industrial noise, but every hearing defect occurring in a worker in a noisy occupation is not caused thereby. There are many causes for depreciated hearing particularly in individuals who are no longer young. As Hallowell Davis[10] says, "There is danger of being made too apprehensive by well-meaning medical warnings and of inviting unwarranted claims for occupational hearing loss"—"We must hope that the medical profession will not base its criterion of a 'significant hearing loss' on audiometric measurements of losses for high tones above speech range. The ability to understand speech is the basic practical criterion of hearing ability and hearing loss. Methods for measuring it directly have been developed and are being improved and a fairly successful attempt to correlate the ability with audiometric measurements has recently been made by a committee of the American Medical Association."

Compensation claims increase in periods of depression and in periods of mass layoffs. Until there is closer cooperation between the industrial physician and the otologist and until a careful pre-employment audiometric hearing examination is made in every worker who may be subjected to excessive

noise, we will not be able to separate the true from the false, the wheat from the chaff. Many workers will receive compensation for a defect which is in no way connected with his occupation, or only partially so. And, vice versa, many a worker whose hearing has been seriously damaged by his occupation will have doubt cast upon his claim. We face the same difficulty in evaluating the claims of demobilized soldiers and sailors because the preinduction hearing examination was so inadequate. This fact is known to all otologists who served in induction centers.

In closing, therefore, my plea to otologists, to industrial physicians, to industry and to insurance carriers is that an accurate pre-employment audiometric examination of the hearing of every worker be made by one qualified to make such an examination and under conditions that are relatively comparable.

BIBLIOGRAPHY.

1. Barr, Thomas: Injurious Effects of Loud Sounds Upon the Hearing. *Brit. Med. Jour.*, p. 675, Sept. 20, 1890. (Quoted by M. H. Taylor, The Laryngoscope, July, 1944.)

2. Horton, G. P.: An Experimental Study of Stimulation Deafness in Guinea Pigs. *Ann. Otol., Rhinol. and Laryngol.*, 44:252, 1935.

3. Crowe, Guild and Polvogt (Quoted by David A. McCoy): The Industrial Noise Hazard. *Arch. Otolaryngol.*, 39:327-330, Apr., 1944.

4. Lurie, M. H.: The Degeneration and Absorption of the Organ of Corti in Animals. *Ann. Otol., Rhinol. and Laryngol.*, p. 712, Sept., 1942.

5. Lurie, M. H.; Davis, H., and Hawkins, J. E.: Acoustic Trauma of the Organ of Corti in the Guinea Pig. The Laryngoscope, Aug., 1944.

6. Lurie, M. H.: The Degeneration and Absorption of the Organ of Corti in Animals. *Ann. Otol., Rhinol. and Laryngol.*, 51:712, Sept., 1942.

7. McCoy, David A.: The Industrial Noise Hazard. *Arch. Otolaryngol.*, 39:327-330, Apr., 1944.

8. Stevens, S. S., and Davis, Hallowell: Hearing, Its Psychology and Physiology. New York, John Wiley & Sons, Inc., 1938. (Quoted by David A. McCoy.)

9. Larsen, B.: Investigations of Professional Deafness in Shipyard and Machine Factory Laborers *Arch. f. Otolaryngol.*, Supp. 36, p. 3, 1939. (Quoted by David A. McCoy.)

10. Bunch, C. C.: Conservation of Hearing in Industry. *Jour. A. M. A.*, 118:588, Feb. 21, 1943.

11. Davis, Hallowell: Abstract from report on "Protection of Workers Against Noise." Ninth Annual Meeting of Industrial Hygiene Foundation at Mellon Institute, Nov. 15, 1944.

12. GARDNER, WARREN H.: Injuries to Hearing. *Indus. Med.*, Sept., 1944.

13. PERLMAN, H. B.: Acoustic Trauma in Man. Clinical and Experimental Studies. *Arch. Otolaryngol.*, 34:429, Sept., 1941.

14. WYDER, CARL G.: Noise in Industry. A Primer of Cause, Effect and Cure. Factory Management and Maintenance Plant Operation Library. No. 69, May, 1944. Johns Manville Co.

15. SABINE, PAUL: The Problem of Industrial Noise. *Am. Jour. Pub. Health*, 34:3, Mar., 1944.

16. TAYLOR, H. MARSHALL: Traumatic Deafness; Problems of Prevention. THE LARYNGOSCOPE, July, 1944.

17. DAVIS, HALLOWELL: Personal communication.

SYMPOSIUM ON NOISE.

(c) UNRECOGNIZED BATTLE NOISE TRAUMA.*

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Many articles have been written about loss of hearing acuity following exposure to noise. One result of these studies is the recognition that some individuals have a greater tolerance for noise than others. Early in the war, the noisiest in history, the authors became interested in the effects of battle noise on the ears of exposed soldiers.

Critical hearing loss accompanying such lesions as ruptured membrana tympani, hemotympani and intracranial injuries were obvious. Our interest focused on the effects of battle noise on the soldier in whom the results of exposure were not so conspicuous. We asked ourselves the question: "Is there any evidence of cochlear injury in the battle exposed soldier who does not complain of hearing loss?" If the answer to this question was affirmative, no matter if only in a small degree, then increased interest should be shown in the protection of the ears in time of battle and simulated battle. It is probable that as much blast or noise is incurred on an artillery range or peacetime maneuver as in actual battle. Certainly in war some must be killed and others maimed, but no effort should be spared to lessen both of these casualties.

To find an answer to our question, audiograms were made on twelve hundred soldiers who had been in active combat, but had no complaint of hearing loss. Although an effort was made to determine duration and degree of exposure to the noise of battle, it was found to be impractical. No individual soldier could compare the intensity of noise in his locality with another, and what one could exaggerate, another could

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minimize. Had an accurate determination of duration and intensity been possible, much might have been added to our knowledge of the effects of noise.

These audiometric studies were made at the 52nd and 55th General Hospitals on soldiers who were casualties of the invasion of Northern France and the subsequent battles. These soldiers were selected at random from the hospital patients who had no auditory complaints. As far as each soldier knew, his hearing was normal. Psychiatric patients naturally were excluded.

To obtain some comparable audiograms for a control series, 150 soldiers from the detachments of the two hospitals were

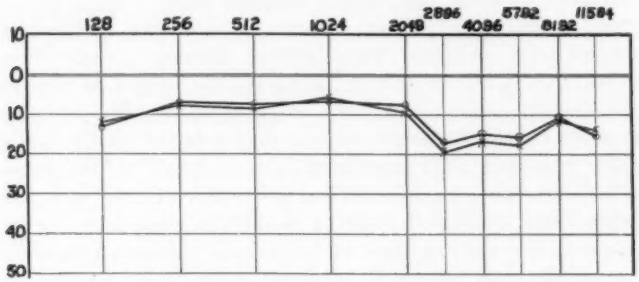

Fig. 1. Composite audiogram of 150 soldiers not exposed to battle noise and with no known loss of hearing. Average age, 26 years.

studied, 100 from one and 50 from the other. These were soldiers permanently assigned to the hospital staff, who had had no exposure to combat, and had no subjective hearing loss. Those with a previous history of ear disease were included provided the hearing was apparently normal. Some were discovered to have varying degrees of hearing loss, but were included inasmuch as this provided a series comparable to the twelve hundred who had no known hearing loss but had been exposed to battle noise. The composite audiogram of the soldiers with no combat exposure is found in Fig. 1.

The composite audiogram of the twelve hundred battle

exposed soldiers appears in Fig. 2. This shows some loss throughout the entire range with the characteristic high tone loss beginning at the 2,896 level. A number of these soldiers stated they had suffered a temporary hearing loss some time during their exposure, but each maintained recovery of this loss by the time of the examination. In most instances it was claimed that the loss was the result of a loud explosion often on one side or the other, usually coincidental with the injury causing their hospitalization. In this group there were 162 who localized this noise on the right, and 158 on the left. A study of the audiograms on these soldiers failed in a goodly percentage to show a correlation between the side of greater

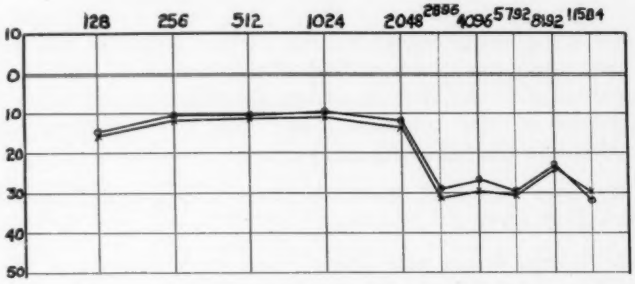

Fig. 2. Composite audiogram of 1,200 battle exposed soldiers. Average age, 26 years.

audiometric loss and the side on which it was maintained the explosion occurred.

An attempt was made to determine the characteristics of the final type of explosion, but this proved to be without value since a lot of men did not know what the type of explosive was; many were wounded by a rifle bullet in a period of comparative quiet after several days of intense noise; some were hospitalized for no wounds (trench foot, etc.), after a period of exposure to noise; and there seemed to be a unanimous disposition among the rest to say "88," since this was the gun which seemed to be the root of all evil. Probably all

this only further proves the unreliability of detailed histories obtained from soldiers concerning the events of combat.

A large percentage of the twelve hundred complained of tinnitus. (No record of the number registering this complaint was kept.) Detailed questioning of this symptom was slighted, as it was desired to avoid a fixation in this regard. When tinnitus was a complaint, audiometric loss of some significance was usually demonstrable.

The records of 1,000 of the 1,200 battle exposed soldiers were studied in greater detail. This number was arbitrarily set in order to make percentages and averages somewhat easier to figure. A reasonably careful history of each of these thousand soldiers was taken and they were categorized in several ways according to the story given. In the light of some of the information obtained, further questions and categories might have been additionally informative. It is to be regretted that the rush of caring for the critically wounded precluded a more intensive study. The 1,000 soldiers were divided into the following categories:

1. Those who were under 30 years of age and those who were 30 or over.

2. Normal drums and drums which gave evidence of adhesive lesions of the middle ear.

3. Exposure before Oct. 1, 1944, and exposure after Oct. 1, 1944.

4. Members of the infantry and members of the armored corps.

5. Hearing loss less than 30 db. in each of the 10 frequencies tested and hearing loss 30 db. or more in any one of the 10 frequencies tested.

CATEGORY 1.

In category 1, it was found that 210 soldiers were 30 years or older and 790 were under that age. Fig. 3 shows the comparative loss between the two groups in this category. Inas-

much as those over 30 averaged about 10 years more in age than those under 30, the difference in the audiometric reading in this instance is hardly significant. This difference may well be laid to the disparity in age rather than a greater susceptibility of the soldiers more than 30 years old. At least there seems to be no sharp evidence that a soldier over 30 is liable to greater audiometric injury in battle than a soldier under that age.

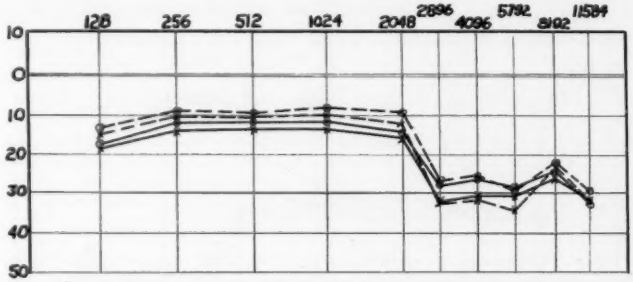

Fig. 3. Category 1. Hearing of battle exposed soldiers under 30 years of age and those of 30 or older.
———— = Before Oct. 1, 1944. — — — — = After Oct. 1, 1944.

CATEGORY 2.

The drums of each of the 1,000 categorized soldiers were studied by means of a Seigle's speculum. These were divided into two groups: those showing normal motility and those with restricted motion. The latter of the two groups gave suggestive evidence that previous inflammatory lesions in the middle ear had resulted in the presence of adhesions. This study was included to ascertain what effect, if any, such adhesions might have upon the transmission of more than normal sound waves. Roughly, two out of 10, or 21 per cent, showed limitation in motion of the drum or malleus. The results of the study may be seen in Fig. 4. There is an appreciable, but not significant, difference between the two groups. Whether or not middle ears with evidence of previous disease are more susceptible to auditory trauma cannot be determined with

accuracy by these figures. One can say that diseased ears are as susceptible as normal ears.

Many eardrums, grossly distorted by previous disease, and with known hearing loss of varying degree prior to exposure to battle noise were also seen. These all met minimal army requirements, but were not included in this study; however, audiograms were made on all those patients who had ever had an audiogram prior to combat. It may be of interest, if enough of these old audiograms can be assembled from private physicians, army records and school examinations to be of significance, to compare them with the recent ones.

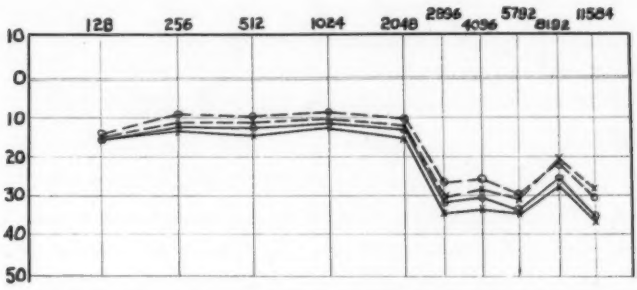

Fig. 4. Fixed drums vs. normal drums. Fixed 217 right and 210 left. Normal 783 right and 790 left.
———— = Fixed drums. — — — — = Normal drums.

CATEGORY 3.

Because there was apparently greater concentration of gun fire in the earlier stages of the European invasion, these same 1,000 soldiers were again divided into those who were exposed before Oct. 1, 1944, and those exposed after that date. The results of this division are depicted in Fig. 5. No appreciable difference can be seen between the two, probably because the cochlear damage sustained in battle is the result of very intense noise of short duration rather than prolonged noise of less intensity. Thus a single shell falling near a soldier in a relatively quiet sector can cause more hearing loss than the many shells of a battle if none fall so near.

CATEGORY 4.

A further division was made. This separated those soldiers who fought with the infantry from those in the armored divisions. A comparison between the two may be seen in Fig. 6. It might well be thought that because of their own fire power and their exposed advanced positions the armored forces would be subject to greater battle noise. The comparison in Fig. 6 shows a slightly greater audiometric loss in the infantry soldiers. This raises the question of the amount of protection to hearing which was given by the wearing of ear-

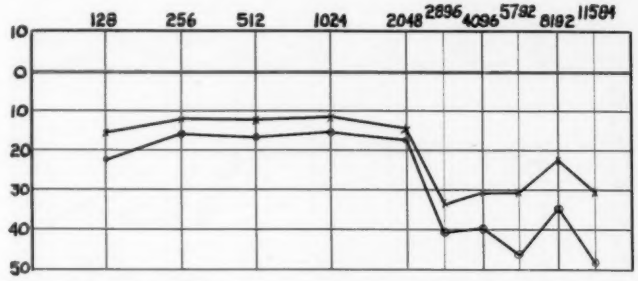

Fig. 5. Exposure before Oct. 1, 1944, and after Oct. 1, 1944. Before Oct. 1, 369. After Oct. 1, 631.

phones and football type helmets with ear flaps by the armored groups. Whatever the decision, the old Army adage that the infantry takes the brunt of battle seems to have further substantiation.

CATEGORY 5.

A final category was made. The thousand soldiers were arbitrarily divided into another two groups. The first includes those whose hearing in both ears was better than 30 db. in each of the 10 frequencies tested, and the second numbers those who had 30 or more db. loss in any one of the 10 frequencies in either ear. This is admittedly an artificial division, but some such separation is necessary to reach an approximation of the number whose ears are affected by bat-

tle noise, for apparently not all who are thus exposed have resultant acoustic trauma. As can be seen in Fig. 7, those included in the first group have an audiometric curve which approximates the curve of the so-called normals in Fig. 1. In a rough way, this separates the normals from those acoustically abnormal. There were 300 who met the requirements of this separation. Thus there were 700 of the 1,000 who showed a loss of 30 or more db. in some one or more of the frequencies tested. In a recapitulation of 100 of the so-called normals it was found that 30 per cent showed a like loss, *i.e.*, a loss of at least 30 db. in one or more frequencies. Or, to

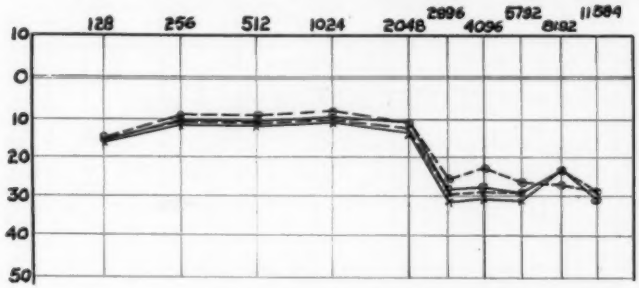

Fig. 6. Infantry vs. armored—infantry, 784; armored, 216.
———— = Infantry. — — — — = Armored.

put it in other words, 30 per cent of an average group of American soldiers not complaining of hearing loss had as much as a 30 db. loss in at least one frequency in their audiometric chart. In group 2 of this last category, 70 per cent of the battle exposed soldiers had a like deficiency in their audiometric reading. Thus among the soldiers in a battle exposed group, there were 40 per cent more who showed this audiometric deficiency than in the average nonexposed group.

In a very rough manner this points out that a very appreciable number of soldiers who have been subjected to battle noise have a recordable degree of acoustic trauma and this approximates 40 per cent of the total. It is only because a

large series was tested that such a statement can be made, for it is obvious that the reason why some positive certain figure cannot be established is because pre-exposure audiograms were not available. In any given soldier it is impos-

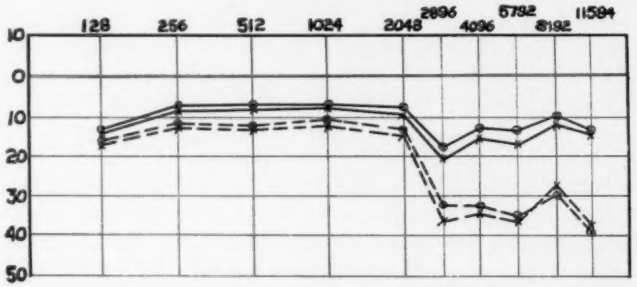

Fig. 7. Audiogram of 300 soldiers with less than 30 db. loss in any frequency, and 700 soldiers with 30 or more db. loss in at least one frequency.
———— = Less than 30 db. — — — — = More than 30 db.

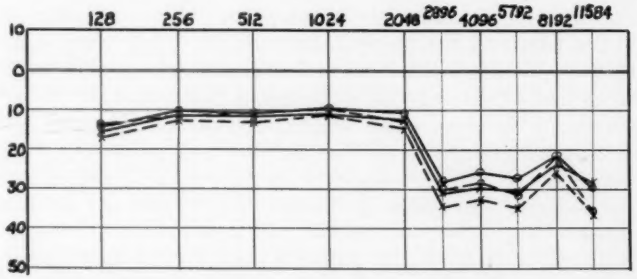

Fig. 8. Audiogram of battle exposed soldiers under 30 years of age compared to those 30 or older.
———— = Under 30 years of age. — — — — = 30 years or older.

sible without an antedating audiogram to state fairly if he is or is not the victim of acoustic trauma from battle noise, for approximately 30 per cent of American soldiers have an appreciable degree of audiometric loss before they enter battle.

This study, it seems to us, offers another reason why, in the American Army, more definite methods of recording the hearing of inducted soldiers should be in force than the present custom of noting only the response to a whisper test. The inadequacy of the latter procedure is widely known, yet it is the standard test of the day. Some change is almost a must if there is going to be any degree of fairness in appraising acoustic compensation claims. At present, to be fair to both the soldier and the government is practically impossible. An interesting incident happened in the hospital of two of us (G. D. H. and S. C. B.). A high ranking officer appeared in the otolaryngologic clinic with a complaint of hearing loss as a result of exposure to battle fire at the Remagen bridgehead. He had the typical audiometric curve of a severely exposed individual with loss in the low frequencies as well as the high. He had marked loss of hearing for speech. An award of the Purple Heart was made and notification of this went to his commanding officer. An official reply questioned the award on the grounds that this soldier had a known severe hearing loss before entrance into battle. Investigation revealed the existence of a pre-exposure audiogram, which was obtained and compared to the postexposure graph. They were almost identical. Without the pre-exposure audiogram, this soldier was almost a certainty to be judged a battle casualty by any otologist. This story deals with a member of an acoustically handicapped group outside the realm of this presentation, but illustrates the problem which may face any otologist when it is necessary to make a decision with pertinent evidence lacking.

Just what significance should be placed on the evidence submitted in this study is difficult at this moment to say. Included in these twelve hundred battle exposed soldiers is a group who have appreciable acoustic trauma, slight though it may be. In none did this damage reach a recognizable level as far as the soldier was aware. None were told of their loss, for it did not fall within the scope of the Army regulations and in any given instance it was impossible to know whether or not the loss existed before combat; but some, according to the figures shown, must have been acoustically

damaged. What effect will this have on the hearing of these individuals five, 10, 20, 30 or 40 years hence? Will the decade by decade advance be speeded to such a degree that handicaps in the speech frequencies will be reached sooner than expected? Or will it make no difference? Dr. Francis Lederer in a personal communication has stated, as a result of his studies with acoustically handicapped sailors at the Philadelphia Naval Hospital, that cochlea degeneration is progressive and audiograms taken a year after trauma was sustained showed a greater degree of loss than the audiogram made just after the initial edema and reaction had subsided. Will minimal losses (compared to such gross changes) show a like tendency? A study of a cross-section of these twelve hundred soldiers five years hence would be most valuable.

All of the above points glaringly to the fact that the armed forces of the United States have not yet adopted protective measures against hearing loss which are effective or inclusive. The pace for the creation of greater destructive power has increased by leaps and bounds since 1918. Protection lags far behind. It seems doubtful if it will ever catch up, but increasing evidence for the need should be a productive prod.

No mention has been made in this paper of the scores of soldiers examined who had severe acoustic damage. Many of them undoubtedly found their way to Hoff, Borden or Deshon General Hospitals. Those who were this fortunate encountered a rehabilitation program which was efficient and effective; but in between these severely handicapped individuals and those with minimal damage as outlined above there are, no one knows how many, thousands who have varying degrees of cochlear damage and through the years will be a problem to themselves, to society and to the otologists of the country. Most of these never reported this handicap; some because they never realized they had it, some because they did not wish to complain of what they considered a natural sequela of their war experience, and many because they feared an examination of hearing would postpone their expected and hoped for discharge from the Army. Still others felt that their hearing difficulty was an ailment from which they

would recover in due season. It is fair to assume that the time will never come when all of these will be sought out and we can know something about the size of the problem which battle noise creates.

There is a fairly simple and obvious conclusion to all of the foregoing statements. It has been pointed out that under present induction examination methods it is not possible to know the acuity of hearing of the soldiers of the armed forces. This study reveals the fact that there must be a very large group of armed personnel with varying degrees of undiscovered or unrecorded battle noise trauma. Faced with these facts, the obvious conclusion is this: lacking the needed evidence, we can never know the extent or the seriousness of the problem of the acoustically handicapped of World War II.

It would seem from this that it is the duty of the otologists of the country to advocate with insistence that the proper measures for acoustical examination of the personnel of the armed forces be provided upon induction and discharge, and that further and continuing studies be made to advance the means of protecting, in time of war, what is one of man's most important senses, the ability to hear.

SYMPOSIUM ON NOISE.

(d) EAR DEFENDERS.*

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Jacksonville, Fla.

The protection of the delicate mechanism of the auditory apparatus has issued a great challenge to medical science. Rarely has such a highly trained group of scientists, both physicists and otologists, expert in their respective fields, cooperated in a more exhaustive study of any phase of preventive medicine.

During the interim between World War I and World War II, the pathology of nerve deafness from acoustic trauma was thoroughly investigated, and research was carried on by many members of this Society. Through these investigations the pathologic changes associated with traumatic deafness are now recognized. With injury, the hair cells of the organ of Corti degenerate and become absorbed. The consequent loss of hearing is permanent. As Lurie[1] has established, the degeneration is not confined to the particular part of the organ of Corti originally damaged; it spreads, and the process may continue for a long period of time.

Though the importance of acoustic insulation was understood, in the interim between World War I and World War II little interest was shown in concerted or concentrated effort to provide adequate means of insulation. At the beginning of World War II, cotton earplugs were advocated for the armed forces, even though it was recognized that cotton has an insulation value varying from 3 to 18 db., while such ear wardens as SMR, MSA, Nelson's Ear Stopper and other commercial defenders have an insulation value ranging from 30 to 40 db.

When World War II had been in progress but a short time,

*Read at the Seventy-eighth Annual Meeting of the American Otological Society, Inc., Chicago, Ill., June 1, 1946.

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the importance of avoiding aural casualties became apparent. Accordingly, research laboratories and centers were then established under the direction of the National Defense Research Committee of the Office of Scientific Research and Development. The major investigations were carried out by the staffs of the Psycho-Acoustic Laboratory, Harvard University; the Department of Physics, University of California at Los Angeles; the Field Artillery Board, Fort Bragg, N. C.; the Army Air Forces School of Aviation Medicine, Randolph Field, Tex.; the Air Technical Service Command, Wright Field, Dayton, Ohio; the Armored Medical Research Laboratory, Ft. Knox, Ky.; the Chemical Warfare Board, Edgewood Arsenal, Md.; the Landing Vehicle Board, Fort Ord, Calif.; and the Medical Research Laboratory, United States Submarine Base, New London, Conn. At these bases the combined talents of the nation's outstanding physicists and otologists were pooled, and investigation of every phase of acoustic insulation was carried on.

The exactions of military requirements demanded a product superior to one intended for ordinary civilian application. Comfort, of equal importance with acoustic insulation, required a material nonirritant, nontoxic, and soft and compliant enough to shape itself to the contours of the auditory canal; also, it needed to be durable and resistant to the effects of ear wax, common cleansing agents and temperature. In addition, in order not to impede communication under conditions of war, a defender was sought which would attenuate all the frequencies of speech to approximately the same extent, to obviate distortion of the speech sounds by the protecting device while at the same time it reduced the whole complex of speech and noise to a level compatible with a reasonable measure of comfort. Likewise, there was the problem of manufacture and distribution, complicated by the necessity of standardization of sizes.

The exhaustive research of this distinguished company of scientists culminated in the development of the ear warden designated as V-51R. Made of black neoprene, a material which meets the requirements mentioned, this defender lends

itself readily to proper shaping, thereby increasing comfort and decreasing leakage. The elliptical body conforms more closely than a round one to the shape of the average ear canal. More flexible than the main body, the bell-shaped flange on the forward end comes in close contact with the wall of the canal; it not only aids in establishing a good seal but also prevents inward travel of the earplug, owing to the pressure

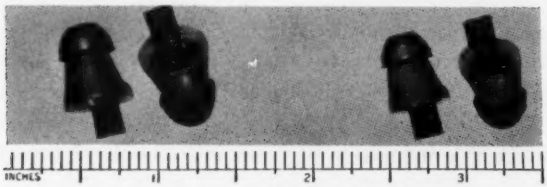

Fig. 1. Photographs of the V-51R Ear Warden.

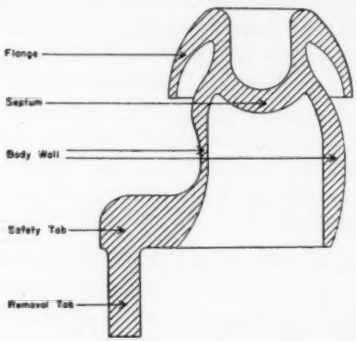

Fig. 2. Cross-section diagram of the V-51R Ear Warden.

wave of a blast or other cause. At the beginning of the hollow stem holding the bell-shaped flange is situated the dome-shaped septum, convex outward, which closes off the stem. The safety tab, perpendicular to the axis of the main body, aids in seating the warden in the canal and prevents inserting it too far. The removal tab facilitates withdrawal.

Obviously, the design of a satisfactory ear defender must

represent a liberal compromise among the three chief factors of amount of insulation against continuous noise, ease of vocal communication and comfort. The V-51R Ear Warden, designed in the nontoxic neoprene form, combines successfully the three essentials of *1.* comfort during many consecutive hours of use by conforming to the shape of the auditory canal and hence applying pressure uniformly over the skin surfaces contacted, *2.* high acoustic insulation over a wide range of audiofrequencies (0 to about 10,000 c.p.s.), and *3.* approximately uniform acoustic insulation over the range of the audiofrequencies, whereby distortionless transmission of speech frequencies at reduced intensities is maintained.

Not only is this device comfortable in most ears, but it affords acoustic insulation of from 25 to 30 db. at the low end and 40 db. or more at the high end of the audiofrequency spectrum. Although originally designed for protection against continuous noise, it also affords adequate protection against the noise and blast of heavy gun fire, being superior to cotton in protecting the wearer against blast effects, as demonstrated in field tests, and it does not interfere with the comprehension of commands. Accordingly, it was recommended that it be standardized for field artillery use. In addition, in the presence of high-level continuous noise, it does not seriously hinder speech communication and may definitely improve it under some conditions.

Industrial application of the V-51R Ear Warden was undertaken by testing it on employees in aircraft factories working under conditions of continuous high-level noise. The evidence appeared to indicate that when adequate instruction is given and the protection afforded by this warden is clearly demonstrated, the employee needing such protection readily avails himself of it. Some officials were of the opinion that its use would appreciably reduce absenteeism, labor turnover and fatigue due to riveting noise.

Actual tests of how well this defender is proportioned were made by fitting it in the three available sizes into 1,191 male ears. Of this number, 19.1 per cent required the small size, 56.7 per cent the medium size, and 24.2 per cent the large

size. Similar data for women showed the need of a smaller size, which was subsequently produced.

Unfortunately, the V-51R Ear Warden was not perfected until the concluding months of the war and was not available even then for use in the combat areas in large quantities. Its superiority over other types of wardens was, however, satisfactorily established. This convenient and comfortable device for occluding the auditory canal, when fitted properly, inserted correctly and used routinely under suitable conditions, should lessen the hazards of temporary or permanent impairment of hearing, both for military personnel and industrial workers. Whether the best possible ear protector has now been made can only be determined by continued research and the lapse of time.

REFERENCE.

1. LURIE, M. H.: War and Deafness. *Conn. State Med. Jour.*, 7:763, Nov., 1943.

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AMERICAN BOARD OF OTOLARYNGOLOGY.

The American Board of Otolaryngology will conduct the following examinations in 1947:

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SYMPOSIUM ON NOISE.

(e) A STUDY OF CASES OF ACOUSTIC TRAUMA FROM A MEDICOLEGAL STANDPOINT, AND A COMPARISON OF PATTERNS OF DEAFNESS.*

EUGENE T. SENSENEY, M.D.,
St. Louis, Mo.

We, as otologists, are often called upon to differentiate between true and fancied acoustic injuries. It must be most confusing to a jury of laymen to hear the contradictory evidence given by physicians who have qualified as experts. The writer believes that certain facts should be generally recognized and established. Then a history of the injury and the symptoms immediately following the injury, a meticulous examination, and a comparison of audiograms of deafness produced by disease with those of deafness due to trauma, will prevent much of this confusion.

The cases to be examined claim deafness caused by fractures of the skull, by concussion from blows on the head, by concussion from blasts or from excessive noise, by concussion from the telephone receiver, caisson deafness and direct penetrating injuries.

In all of these cases the acoustic insult is immediate. In most instances the hearing improves following the injury; in some it remains the same, in a few it becomes worse. In the last category belong those cases with a rupture of the eardrum, or a penetrating wound of the eardrum, with subsequent infection; those cases with so profound a loss that the remnant of hearing fails from nonuse,[1] or from "the accompanying degeneration of the nerve fibres and ganglion cells leading from the site of the lesion";[2] those cases with latent syphilis;[3] and those cases with dislocation of the ossicles with subsequent scar fixation.

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*Read as part of a Symposium on Noise at the Seventy-eighth Annual Meeting of the American Otological Society, Inc., Chicago, June 1, 1946.

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From whatever cause, we repeat that the first insult to hearing is immediate and not weeks or months after the injury. Although one may become deaf gradually over a period of time without being aware of the hearing loss, sudden deafness is very annoying. One has only to watch a swimmer jumping up and down and banging his head to get rid of "water in his ear" to realize the truth of this statement. Some of those injured are unconscious for a period after the injury, but we have yet to see one with a real injury who did not realize the deafness soon after regaining consciousness. We believe this to be the most important from a medicolegal standpoint. The plaintiff who states that his deafness was first noticed months after a trauma has no proper claim.

Coincident with acoustic insult are tinnitus aurium, vertigo and disturbances of equilibrium. Indeed, in most cases of acoustic trauma we have a triad: deafness, tinnitus, vertigo. The tinnitus may persist and may be most annoying. Those suffering from it deserve some compensation. (Fowler[4] has suggested a method of measurement of tinnitus: "One applies a sound similar to the tinnitus in the opposite ear, and balances it in loudness with the tinnitus.") Vertigo is usual after injuries. As a rule, it disappears after a few weeks; however, it may reappear years after the injury. Baumoel and Marks[5] consider these late manifestations as "functional" and believe them to be circulatory; and that "the brain stem is frequently involved with the rest of the brain in cases of head injuries producing the clinical picture of concussion." It should be noted that in the cases they report, violent symptome occurred at the time of the accident.

As dizziness is a common symptom, the individual complaining of it following a trauma should have an exhaustive examination of the vestibular apparatus. Indeed, the end-result may represent a far greater disability than the loss of hearing. The following case is an example.

This man, age 40 years, was struck by an automobile. In addition to other injuries, the X-ray showed a fracture of the skull, extending into the right petrous bone. He was unconscious for 10 days. We saw him on the twelfth day. He complained of severe headache, tinnitus and dizziness, and was completely deaf in the right ear. There was spontaneous nystagmus. Two months later, the dizziness had disappeared. The deaf-

ness was total in the right ear. There was no response to the caloric test on the right side, but after-turning nystagmus showed a duration of six seconds on both sides. A year later he returned, complaining once more of dizziness and staggering gait. The duration of the after-turning nystagmus was only one second on both sides. His condition was truly pitiable. He lived in constant terror of these attacks of vertigo and had had a number of bad falls. Spontaneous nystagmus could be elicited by positional changes While he had received compensation for his total unilateral deafness, the latter was the least of his troubles.

There seemed to be no legal way in which his case could be reopened. Ruttin,[1] to whom the writer described this case, stated that in total destruction of one labyrinth, while compensation might follow for a while, degeneration often took place in the opposite labyrinth later.

TRAUMATIC INJURIES OF THE MEMBRANA TYMPANI.

Traumatic injuries of the drum membrane, whether due to rupture by compression of air within the canal or by direct puncture of the membrane, rarely *per se* cause much impairment of hearing. Indeed, in "blast" injuries, since the synergistic protective reflex of the stapedius and tensor tympani muscle is only momentary, "the degree of deafness is usually greater when the tympanic membrane remains intact."[6] The immediate symptoms of rupture or puncture of the drum are severe and startling; loud noise, severe pain, great tinnitus, vertigo, falling, staggering gait and stupor, but only slight deafness. These symptoms, except for the tinnitus, disappear rapidly. The tinnitus may persist for a time. If, however, there follows a secondary suppurative process in the middle ear, or if the ossicles are dislocated, profound deafness may result. Such deafness is due to the formation of adhesions and granulations, and the picture is that of conduction deafness: Weber to the injured side, Rinne negative, and the deafness more pronounced in the lower frequencies. If a simultaneous concussion of the labyrinth has taken place, the sound of a tuning fork placed on the vortex is lateralized to the nonaffected ear, the Rinne test positive, and the loss of hearing is greater in the higher frequencies.[7]

A traumatic injury is readily recognized in the first days following the injury. The margins of the perforation are

sharply defined and are covered here and there with coagulated blood. If seen later, after a secondary infection has taken place, the differentiation between a rupture with subsequent infection and a suppurative otitis media is impossible from a medicolegal standpoint.

Case 1: This young man, a helper at a gun club, endeavored to assemble a 12-gauge automatic shotgun, having first placed a shell in the barrel. Following the explosion close to his left ear, he experienced pain in his left ear, tinnitus and severe vertigo. Five hours later, these symptoms, except for slight tinnitus, had disappeared. The left eardrum showed a jagged perforation, the margins of which were covered with blood. His hearing was only slightly affected. The perforation healed rapidly, his hearing became normal, and he entered the Navy two months later.

Case 2: This young man, age 20 years, was employed on a large farm in Illinois. While engaged in clearing some land of "buck-brush," a twig of this tough fibrous wood entered his right auditory canal. He experienced great pain, dizziness, tinnitus and deafness. The pain increased. A few days later, when we examined him, suppuration was well established. The malleus was dislocated, the membrana tympani perforated, with many fine wood fibres in the wound. Rinne negative, Weber to right. He returned after a month. The entire middle ear was a mass of granulations, studded with wood fibres. The sound conduction deafness was profound. (Operative procedure was refused.)

The two cases which follow are most interesting. The injuries were identical, as were the symptoms.

Case 3: This man, age 28 years, was employed as a checker in a trucking company. While helping to unload a heavy box from a truck, the box fell on him, forcing a slender, flat marking pencil, which he carried in his shirt pocket, into his left external canal and through the left membrana tympani. The pain was severe, as was the vertigo. He was nauseated. He could not stand erect, falling always forward on his face. These symptoms disappeared quickly. While eating his luncheon, he noticed a loss of taste on the left side of his tongue. Examination a few hours after the injury showed a puncture of the membrana tympani, extending from just posterior to the umbo of the malleus upward through the posterior fold. His hearing was normal, except in the higher frequencies. Rinne positive, Weber equal. The wound healed rapidly. Seen one month later, the hearing was normal for all frequencies and his sense of taste had returned.

Case 4: This woman, age 39 years, ran a steel tape needle (flat botkin) into her left ear. She complained of excessive tinnitus and dizziness, was unable to stand erect, "falling backwards and forwards, so that the girls thought she was putting on." These symptoms disappeared quickly. She noticed that she had lost the sense of taste on the left side of her tongue. Examination made on the same day showed a puncture of the membrana tympani, posterior to the handle of the malleus, extending through the posterior fold. The wound healed rapidly without suppuration or the formation of adhesions. She returned some months later, complaining of total loss of hearing in the left ear. By the use of tests for simulation, we were able to prove that she could hear conversational speech in the left ear.

TELEPHONE DEAFNESS.

We have had no cases of trauma while using the telephone. Many cases have been reported in the literature. In most of these, a loud click has been followed by fullness in the ear, dizziness and tinnitus, with or without a rupture of the drum membrane. In most cases the deafness, if present, cleared up

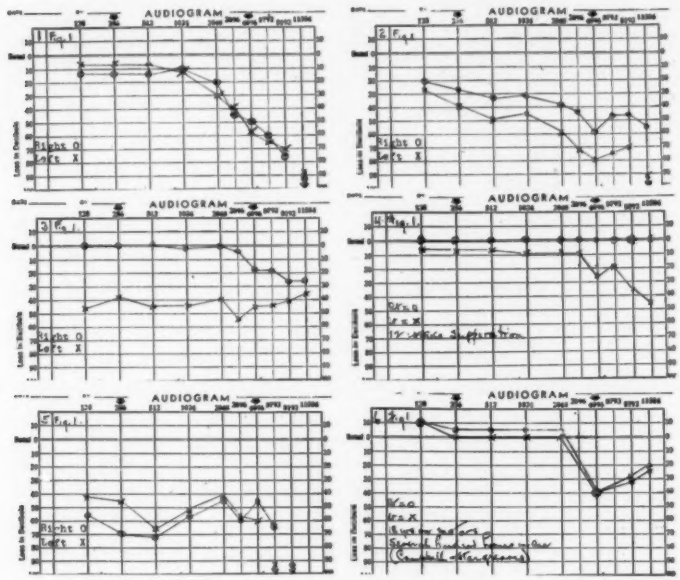

Fig. 1.

Graph 1. Male, age 70, in excellent health, has begun to notice some deafness. Pattern typical of presbycusis.

Graph 2. Male, age 43, consulted us for deafness. Has had a great deal of trouble with his teeth. Deafness resulting from focal infection.

Graph 3. Male, age 40, has had a purulent discharge from his left ear for many years. Pattern of conduction deafness (plus some nerve degeneration in both ears).

Graph 4. Male, age 16, had acute suppuration in left ear of 12 weeks' duration. Operation was refused. Chief loss in higher frequencies.

Graph 5. Male, age 35, attributed his deafness to an acute bilateral otitis media in childhood. Both eardrums are normal. The loss of hearing for lower frequencies, prolonged bone conduction and negative Rinne (Bezold's triad) inclines us to believe that an otosclerosis has been superimposed on any deafness left by his acute otitis media. History of familial deafness. Two brothers are deaf.

Graph 6. Male, age 40, after working on motors for 18 years. Had several hundred hours in air. (Campbell-Hargreaves[11])

rapidly. Bunch[5] reported a case of a young girl with severe permanent deafness, following the use of the telephone when the wire was struck by lightning (see Fig. 2, Graph 4). His other cases conform to patterns of occupational deafness.

CAISSON DEAFNESS.

We have had but one case of caisson deafness. This man,

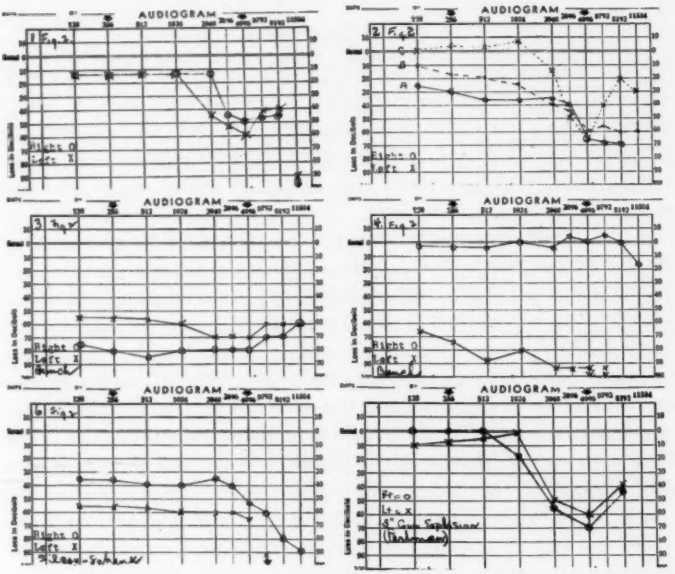

Fig. 2.

Graph 1. Male, age 52, was referred by his wife who claimed he was getting deaf. His audiogram shows the tonal dip at 4,096 d.v., established by Bunch for occupational deafness. Inquiry showed that, as a railroad man, he had spent years traveling on the trains.

Graph 2. The audiograms of three patients who have a much greater loss for tones of high pitch than for low tones. "A" is the record of a physician, age 50 years, whose deafness has been progressive for more than four years. "B" is the record of a woman, age 67 years. This is interpreted as being an example of presbycusis. "C" is the record of a man, age 30 years, who has been subjected to loud noises from various sources. (Bunch.[5])

Graph 3. Male, age 35 years, caisson deafness. (Bunch.[5])

Graph 4. Female, age 12, showing profound deafness after lightning struck telephone wire while she was using telephone. (Bunch.[5])

Graph 5. Male, age 22, made two weeks after a motor shell explosion 10 feet away. Both tympanic membranes ruptured, bilateral aural discharge. (Silcox-Schenck.[6])

Graph 6. Male, deafness due to 5-inch gun explosion. (Perlman.[8])

age 40 years, following too rapid decompression, suffered tinnitus and dizziness and vomited. He was deafened in both ears. All symptoms disappeared quickly, and the hearing returned to normal in a few weeks. Bunch[8] has shown that in severe cases the hearing loss is uniform throughout the range of the audiometer (see Fig. 2, Graph 3). Whether the deafness is temporary or permanent, it becomes manifest an hour or so after the decompression.

BLAST DEAFNESS.

The writer has had no cases of blast deafness. Silcox-Schenck[9] recently have described 82 cases seen in the Navy. They state that, "in the majority of the audiograms of patients subjected to blast, the greatest loss was in the higher frequencies, but the tonal dip at 4,096 cycles seen so commonly in cochlear damage due to industrial noises and repeated excessive sound stimuli was not evident in many of these audiograms. There was a relatively larger decibel loss in patients with secondary infection, due to the associated impairment of conduction." Owing to the exigencies of war, their audiograms were made under unfavorable conditions and, in our opinion, much too soon after the injury (two to six weeks) to judge the final result. One of their audiograms is reproduced in Fig. 2, Graph 5. More characteristic is an audiogram by Perlman[9] of deafness following a five-inch gun explosion.

OCCUPATIONAL DEAFNESS.

While we, as experts, are called upon only occasionally to testify in cases of slowly progressing deafness, it is well that we understand them in order to evaluate accidents. Crowe, *et al.*,[10] have called the curves found in progressive deafness "gradual high tone loss" and refer to the sudden drops so often seen in the residual deafness following acoustic insult as "abrupt high tone loss." We reproduce in Fig. 2, Graph 2, a combined audiogram of Bunch,[8] showing this difference extremely well.

Bunch,[8] after a most exhaustive study, came to the follow-

ing conclusion: "If a loss of auditory acuity has resulted from the effects of excessive stimulation of industrial noises, it is first evidenced by an abrupt dip or gap in the hearing range as determined by the audiometer, usually near c_5 (4,096 dv.). Acoustic trauma should be suspected when these gaps appear. They are very commonly found in the records of the hearing of sportsmen and hunters who often have no loss in acuity for the spoken voice and are unaware of any hearing defect whatsoever.

"With continued excessive stimulation, this gap in the curve becomes deeper and broader, to include the tones above and below c_5. Eventually, the extremely high tones of the audiometer become inaudible and the curve shows an abrupt drop, usually between c_4 and c_5. Many times there is a tendency for the curves to approach the normal lines at c_6, with the deepest point in the gap near c_5."

Many observers have noted this dip at 4,096 dv. Campbell and Hargreaves,[11] describing aviation deafness, believe this to be due to the position of the cochlear representation of the 4,096 area and its greater exposure to the sounds produced by flight. Larsen[12] thought that this especial vulnerability might be due to the poor blood supply of this area. He calls attention to the occurrence of this circumscribed defect following head injuries. Grove[13] states that the deafness from concussion of the labyrinth is of the perceptive type and is "undoubtedly due to hemorrhage into the basal coil of the cochlea." The experiments of Perlman[9] and of Davis, *et al.*,[14] tend to prove that the location of the chief loss of hearing depends on the spectrum of the noise to which the ear is subjected.

"Practically all Workmen's Compensation Acts are founded upon the theory that an 'accident' or 'occupation disease' must be on the basis of compensable disability. Much has been written on the definition of these terms and the relationship between them. Hardly anywhere in the field of industrial hazards does the distinction between the two grow so faint as in cases involving sight and hearing losses. An occu-

pational 'disease' has been defined by the Missouri Supreme Court to be:

> "'. . . a disease contracted in the usual and ordinary course of events, which, from the common experience of humanity is known to be incidental to a particular employment.'

"On the other hand an *'accident'* is defined as:

> "'An unexpected and unforeseen event happening suddenly and violently, with or without human fault and producing at the time objective symptoms of an injury.'

"The legal distinction between a disease and injury resulting from a series of accidents may be abstractly quite clear, but the application of the rule stating that distinction is sometimes quite difficult from a medical standpoint. Any necessity for such distinction cannot be fully appreciated by the medical profession. More accurately the basic fact to be ultimately determined would seem to the physician to be whether the loss of function has resulted from the patient's occupation, or otherwise. If it has, it would seem to be one for which he should clearly be compensated so far as permissible, and possible, under the applicable legal rule. Actually the courts have been somewhat divided on the question, although liberality in applying the test seems to be more often the rule than the exception.

"In North Dakota, for example, compensation was awarded a telephone operator for six months' period of disability due to the fact that 'she received in her ears at various times intense vibrations, static, loud noises and ringing bells.'

"Similarly, in an Oklahoma case, an employee was awarded compensation for partial loss of hearing resulting from his close proximity to the exhaust of a pump in the oil fields. The medical testimony used as a basis for the award was to the effect that 'vibrations of the exhaust, loud noise would more or less destroy the hearing.' The award was made on the theory that the employee had suffered an 'accident,' rather than an 'occupational disease' since the condition could

be 'referred to some definite event, the date of which can be fixed with certainty, but which cannot be fixed in the case of occupational diseases'."[15]

It will be seen from above that those employed in noisy occupations, who become deaf gradually, often receive no compensation unless a specific accident or a series of specific accidents can be pointed out.

According to Larsen,[12] certain countries prior to the war had insurance laws which included occupational deafness. In Germany, deafness did not become invalidating until a person was unable to hear conversational speech at a distance of 1 m. with the better hearing ear. Of the 250 workers examined by Larsen, only five fell in this classification, and all five continued at work receiving full wages.

The human ear was never intended to withstand the noise of modern industry. That some are more susceptible than others is definitely known. Examination of hearing at the time of employment is usually perfunctory or nil. (A careful examination would protect the employer from professional malingerers). No periodic examinations are made and rarely are safeguards provided. The only therapy of occupational deafness is change of occupation or the establishment of sufficient rest periods free from noise. Both are impracticable. Prophylaxis remains; namely, to reduce the noise of industry (*e.g.*, substitution of welding for riveting), and to provide the workers with effective ear wardens.

DEAFNESS FROM INJURIES TO THE HEAD.

Politzer[7] stated that "a medicolegal decision as to the existence of concussion of the labyrinth can be given only in those cases in which there is a fissure of the temporal bone extending to the external meatus, and in which an injury of the labyrinth can be inferred, either from the discharge of cerebrospinal fluid or from complete deafness and the absence of perception through the cranial bones. Those concussions of the internal ear produced either by direct action of violence to the head or by detonations, in which the external meatus

and membrana tympani present a normal appearance, are absolutely incapable of being judged from a medicolegal standpoint. The reasons for this are the following: *1.* Proof cannot be given that the paralysis of the auditory nerve is due to the presumed injury; and *2.* even if the traumatic violence has been established, it cannot be positively determined whether the paralysis of the acoustic nerve had not already existed before receiving the injury."

This opinion has caused much controversy through the years. The writer believes it to be true only in part, and does not think this statement would have been made if Politzer had possessed proper X-ray facilities and an audiometer. When a fracture extends through the otic capsule, one may expect total unilateral loss of hearing; however, the writer has seen cases of longitudinal fracture splitting out through the tympanum and the roof of the external auditory canal, with the escape of cerebrospinal fluid, where very useful hearing remained. Grove[16] has reported many such cases, with varying degrees in the loss of hearing. Such injuries should be considered concussions of the labyrinth. The hearing loss is perceptive in type, except where some derangement has taken place in the middle ear, and conduction deafness is added.

The writer believes that in the case of head injury, with the history of violent symptoms of labyrinthian disturbance, lessened response to vestibular tests and an audiogram showing perceptive hearing loss, the pattern of which does not conform to those of perception deafness caused by disease, one may state with assurance that the hearing loss has been caused by the accident.

In concussions of lesser degree, while the symptoms are immediate, recovery is more rapid and more complete. Examined after a lapse of months, the vestibular reactions may be normal. Tinnitus may persist. The hearing loss is chiefly in the higher frequencies, and the pattern closely conforms to those of deafness caused by noise. Unless by careful inquiry we can eliminate exposure to noise, we can make no definite statement.

Most of the cases of acoustic trauma seen by the writer have been individuals who have received blows on the head. The deafness is usually on the side of the head receiving the blow; however, if the individual is knocked down, the opposite side of the head may receive the greater impact. From our records we have selected the following cases.

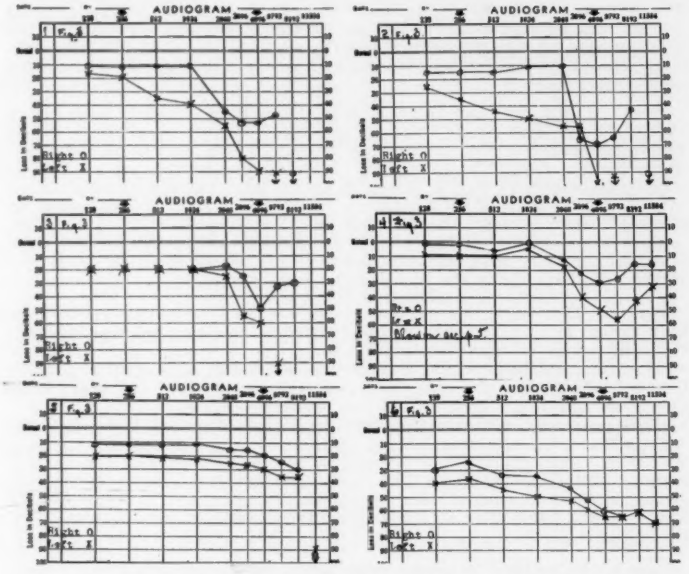

Fig. 3.

Graph 1. Male, age 49 years. Fracture without fissure into petrous bone. Blow on left side of head. Audiogram shows profound disturbance. No frequencies above 4,096 d.v. heard on left side. Consider this true injury.

Graph 2. Male, age 41 years. Blow on left side of head. No demonstrable fracture. No frequencies above 4,096 d.v. heard on left side. Consider this true injury.

Graph 3. Male, age 44 years. Blow on left side of head. Symptoms not so severe as in preceding cases. Failure to hear frequencies above 4,096 d.v. inclines us to believe that his is a true injury. No history of exposure to excessive noise.

Graph 4. Male, age 16 years, blow on occiput. Epileptic. No history of exposure to noise.

Graph 5. Male, age 56 years. Blow on left side of head. Audiogram is typical of gradual high tone loss. We believe that his deafness existed prior to the injury.

Graph 6. Male, age 67 years. Blow on right side of head. Audiogram typical of focal infection. Claimed loss of hearing on right side. It will be noticed that his hearing is better on the right than the left side. It was learned later that he had used his deafness in a previous damage suit. Had chronic arthritis and very bad teeth.

Case 1: This man, age 49 years, was hit by a streetcar. He was unconscious for six days. There was "an extensive fracture of the skull involving both posterior parietal regions, especially the left, and extending down into the occipital region and forward into the right side of the occipital bone. There was a separation of the left mastoid suture and anterior portion of the lambdoid suture and also a separation of the posterior portion of the sagittal suture. The fragments on both sides are separated, but this was especially marked on the left." He bled from the left ear. Upon regaining consciousness, he noticed deafness in his left ear with great tinnitus, but no vertigo. The audiogram is shown in Fig. 3, Graph 1. In both ears there is a marked dip beginning at 1,024 dv. and the maximum is reached at 4,096 dv. We consider that he had a concussion of both labyrinths, more marked on the left. Caloric reaction present on both sides, but feeble. Examination made six months after injury.

Case 2: This man, age 41 years, was struck by a streetcar with injury to his left occipital region. He was unconscious 10 or more hours. Upon regaining consciousness, he vomited repeatedly and complained of headache, vertigo, tinnitus and deafness (more marked on the left side). The audiogram shown in Fig. 3, Graph 2, shows the right ear to be approximately normal up to 2,048 dv., then a tonal dip reaching its maximum at 4,096 dv., then a rise. The left ear shows a gradual drift downwards, with a great dip at 4,096 dv. and no frequencies heard beyond this. Hypoirritability of vestibular apparatus. This seems to us to be a true injury. Examination made five months after injury.

Case 3: This man, age 44 years, was hit by a streetcar, received a blow on the left side of head and left shoulder. He was not unconscious. He complained of immediate deafness in the left ear, severe tinnitus and vertigo. Audiogram, made eight months later, is shown in Fig. 3, Graph 3. Here the low tones are relatively unaffected, but there is a sharp dip beginning at 2,048 dv. and frequencies higher than 4,096 dv. are not perceived in the left ear. He still complained of dizziness. His description of his dizziness did not conform to that of vestibular vertigo. We were unable to produce nystagmus by changes in position. After-turning nystagmus equal on both sides and of good amplitude, lasting 16 seconds. We consider this a borderline case, but in view of the complete loss of hearing in frequencies above 4,096 dv., we consider this a true injury.

Case 4: Male, aged 16 years, was caught between two moving streetcars and badly mauled. A chip of bone was knocked off his external occipital protuberance, and the left side of his head and face was badly contused. He complained of severe tinnitus, but no aural vertigo. He was noticeably deaf in the left ear immediately following the accident. At the time of our examination, made some months later, he had no tinnitus or vertigo and did not think he was deaf. Audiogram is shown in Fig. 3, Graph 4. It is interesting to note how closely this pattern follows those of noise deafness. The boy was an epileptic, and had been kept from firearms and the like. Careful inquiry showed no exposure to loud noise at any time in his life.

In the cases that follow, while the histories are similar to Cases 1, 2, 3 and 4, the audiograms show that there was no lasting effect on the hearing from the blows on the head. The writer believes that in most such cases probably a slowly

progressing loss of hearing has been unnoticed until a mild concussion called attention to the existing deafness.

Case 5: This man, age 56 years, received a blow on the left side of his head in a streetcar accident. He bled from his left ear. He was unconscious for a short period. He compained of tinnitus, vertigo and deafness in the left ear. When examined eight months later, the tinnitus and vertigo had disappeared, but he claimed deafness in the left ear. The audiogram shows a beginning presbycusis, the hearing in the left ear slightly poorer than in the right. Both membrana tympani show slight retraction (see Fig. 3, Graph 5). No lasting injury.

Case 6: This man, age 67 years, was struck on the right side of the head with a heavy suitcase, which fell from the luggage rack of a train. He experenced immediate tinnitus, but no vertigo. He states that his hearing was perfect in both ears prior to the accident. He now (one year following the accident) claims that the hearing in his right ear is impaired, but that he hears normally in his left ear. The audiograms are typical of a gradual progressing deafness in both ears. The hearing in the left, or supposedly good ear, is an average 10 db. less than in the right ear. A verdict was rendered for the defendant. Later it was discovered that he had used his hearing defect as an allegation in a previous damage suit (see Fig. 3, Graph 6).

PATTERNS OF DEAFNESS.

The comparison of the audiograms of plaintiffs alleging deafness with the graphs of deafness caused by aural disease, or of known trauma, is of great value in determining the justice of claims. The audiograms shown in Figs 1, 2 and 3 are from the writer's records and from those of Bunch,[8] Silcox-Schenck,[9] Perlman[9] and Campbell-Hargreaves.[11]

The patterns of gradual high tone loss, whether from focal infection or from old age, are very definite. The claim of deafness from an accident of any plaintiff whose audiogram conforms to these patterns must be regarded with suspicion. The graphs of deafness from chronic suppuration and otosclerosis do not differ greatly. The examination of the tympanic membranes and tuning fork tests are necessary for differentiation.

The audiograms of Cases 1, 2 and 3 (see Fig. 3) seem to the writer to show most graphically that a violent disturbance of the perceptive apparatus has taken place. There is a more or less rapid drop to 4,096 dv., and no higher frequencies are heard beyond that point on the side receiving the maximum force of the blow. In this, these curves differ from

those of industrial deafness (Bunch[2]) where, after the tonal dip at 4,096 dv., the higher frequencies are heard. Cases 5 and 6 are typical of gradual high tone loss, and we do not believe that the head injury had anything to do with the loss of hearing. Graphs of hearing loss from blows on the head may closely resemble those due to noise. Hence, a careful inquiry should be made into the past history of those claiming deafness from head injuries.

CONCLUSIONS.

1. In any acute acoustic trauma producing deafness, the impairment of hearing is immediate. The deafness usually is associated with vertigo and tinnitus. These symptoms, if present, are also immediate.

2. Vertigo may be a greater economic disability than loss of hearing. In cases complaining of dizziness, the vestibular apparatus should be carefully investigated. One should hesitate before giving too favorable prognosis. Tests should be made for latent nystagmus, and for hyperirritability, hypoirritability and for imbalance.

3. Audiograms are useful in differentiating between true injuries to hearing and false claims. To be of value, these graphs should be made meticulously, with due attention paid to possible simulation on the part of the plaintiff. They should be made in a soundproof room or in one where the masking by extraneous noise has been carefully calibrated. Tuning fork tests should always be used in conjunction.

4. In occupational deafness, compensation awards seem unsatisfactory. Insurance plans fail because the pension for invalidating deafness never approaches the sum that the worker earns while at work. The only answer is prophylaxis, prevention of deafness, whether this be accomplished by education of industry, or, if necessary, by legislation.

REFERENCES.

1. Ruttin, E.: Communication.
2. Lurie, M. H.; Davis, H., and Hawkins, J. E., Jr.: Acoustic Trauma

of the Organ of Corti in the Guinea Pig. THE LARYNGOSCOPE, LIV:8, Aug., 1944.

3. BRUNNER, H.: Communication.

4. FOWLER, E. P.: Head Noises in Normal and Disordered Ears; Significance, Measurement, Differentiation and Treatment. *Arch. f. Otolaryngol.*, 39:6, June, 1944.

5. BAUMCEL, S., and MARKS, M. S.: Evaluation of Vertigo Following Head Injuries. *Arch. Otolaryngol.*, 33:2, Feb., 1941.

6. SILCOX, LOUIS E., and SCHENCK, H. P.: Blast Injury of the Ears. *Arch. f. Otolaryngol.*, 39:5, May, 1944.

7. POLITZER, A.: Diseases of the Ear. Lea Bros. & Co.

8. BUNCH, C. C.: The Diagnosis of Occupational Deafness; an Historical and Audiometric Study. THE LARYNGOSCOPE, XLVII:9, Sept., 1937.

9. PERLMAN, H. B.: Acoustic Trauma in Man. *Arch. f. Otolaryngol.*, 34:3, Sept., 1941.

10. CROWE, S. J.; GUILD, S. R., and POLVOGT, L. M.: Observations on the Pathology of High Tone Deafness. *Bull. Johns Hopkins Hosp.*, LIV, 1934.

11. CAMPBELL, P. A., and HARGREAVES, JOHN: Aviation Deafness. *Arch. f. Otolaryngol.*, 32:3, Sept., 1940.

12. LARSEN, B.: Investigation of Professional Deafness in Shipyard and Machine Factory Laborers. *Acta Otolaryngol.*, Supp. 36, 1939.

13. GROVE, W. E.: Discussion of Unilateral Deafness (Hughson). *Trans. Am. Otol. Soc., Inc.*, XXXIII, 1944.

14. DAVIS, H.; MORGAN C. T.; HAWKINS, J. E., JR.; GALAMBOS, R., and SMITH, F. W.: Temporary Deafness Following Exposure to Loud Tones and Noise. THE LARYNGOSCOPE, LVI:1, Jan., 1946.

15. GUNN, MR. DONALD: Communication.

16. GROVE, W. E.: Skull Fractures Involving the Ear. THE LARYNGOSCOPE, XLIX:8-9, Aug.-Sept., 1939.

In addition to the references listed above, the writer acknowledges the help of S. R. Silverman, Ph.D., who has been a constant source of information.

COURSE IN OTOLARYNGOLOGY, RHINOLOGY AND LARYNGOLOGY.

The Eleventh Annual Graduate Convention in Otology, Rhinology and Laryngology will be held at the University of Cincinnati College of Medicine, April 14 to April 19, inclusive, 1947. This is to be an operative course on the cadaver and didactic lectures.

BOOK REVIEWS.

Clinical Methods of Neuro-Ophthalmologic Examination. By Alfred Kestenbaum, M.D. First edition, clothbound, 383 pages. Grune & Stratton, New York, 1946. Price $6.75.

The author states that this book is primarily intended to present a review of the clinical methods of ophthalmologic examination that may be helpful in neurologic differential diagnosis. This book has presented the subject matter briefly, yet as accurately, as completely as could be expected in a small volume. An extremely large number of tests have been described. No effort has been made to state their relative value or which ones are used more extensively at the present time; that is left to the discretion of the reader. The first five chapters dealing with visual pathways appear to be the most valuable contributions. The anatomy is particularly good; however, its brevity requires that the reader know a considerable amount of neuroanatomy or else have such texts close at hand for reference.

This little volume may serve as a review of the subject matter pertaining to neuro-ophthalmology. It does not serve as a reference book on the subject of neuro-ophthalmology, nor is it a book intended to present neuro-ophthalmology as a clinical treatise to the beginner.

M. J. R.

The Etiology of Deaf Mutism, with Special Reference to Heredity, by Harald Lindenov (translated from the Danish by Axel Andersen), J. Jorgensen & Company, Copenhagen, 262 pages.*

Lindenov's study at the University Institute for Human Genetics, Copenhagen, had as its aims:

1. To examine whether an objectively clinical clue can be found to the differential diagnosis between hereditary and non-hereditary deaf mutism.

2. To examine whether, through an objective, clinical examination of the hearing children of the deaf mute—possibly other relatives—some clue may be found that will disclose a taint or disposition to deaf mutism.

3. To state the numeric proportion between persons with hereditary and those with nonhereditary deaf mutism, and, in the various subsections of these two main groups, to indicate the sex distribution and the importance of consanguinity.

4. To examine the importance of external influence to the occurrence of deaf mutism.

5. To find the inheritance of the hereditary cases.

6. To examine whether any disposition to acquired deaf mutism may be demonstrated and, if so, how it manifests itself.

7. To find out whether the various other disorders of possible hereditary determination that occur in association with deaf mutism or in the families of the deaf mutes have any relation to deaf mutism and, if so, which relation.

8. To state the importance of the results arrived at, with a view to eugenics.

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*Reprinted from the Volta Review, December, 1946.

The study involved personal examinations of 32 families with deaf mutes and a census examination of deaf mutes in certain Danish communities. Of 678 deaf mutes employed, 480 appropriately classified nonselected individuals were examined. This group is an excellent representation of the deaf population in the communities under consideration.

The first six chapters of the monograph are introductory and furnish a comprehensive review of the pertinent literature. It is of interest to Americans that Lindenov, in tracing the literature on deafness from Herodotus to Urbanschitsch, Denker and Fisher, takes issue with Fay's (Marriages of the Deaf) findings. He criticizes Fay's report on the grounds of *1.* validity and reliability of the questionnaire technique, *2.* Fay's competence as a layman in genetic and medical matters, *3.* the lack of general acceptance at that time (1898) of Mendelian theories of heredity.

We might take issue with Lindenov's classification as a deaf mute a child whose onset of deafness occurs at or prior to seven to eight years of age. This is too advanced an age to warrant such classification. He quite properly cautions us not to accept feeble-mindedness and aphasia as deaf mutism. Lindenov disposes of rotation and galvanic reaction tests as inadequate for satisfactory differential diagnosis of deafness. Evidence seems to indicate that Muck's adrenalin probe reflex, Langenbek's principles of symmetry and X-ray examination have definitive diagnostic value. In general, past investigations indicate that hereditary deaf mutism may be characterized by symmetrical remnants of hearing and acquired deaf mutism sows an asymmetrical pattern of hearing residue. Furthermore, hereditary deaf mutism shows irritability of the vestibular apparatus while it is either decreased or nonexistent in persons with acquired deaf mutism. The relationship of the structure of the temporal bone and irritability of the sympathetic to heredity deafness are also discussed.

Lindenov discusses the relationship to deaf mutism of other disorders such as retinitis pigmentosa, feeble-mindedness and ataxia, and presents evidence of mild correlations with these entities. He questions Hammerschlag's theory of a common organic origin for sporadic deaf mutism (degenerative type such as from consanguinity), heredolabyrinthic deaf mutism (local disorders of organ of hearing), and otosclerosis.

The literature suggests many hereditary theories and the general conclusion is that sporadic deaf mutism reveals a monomeric, recessive inheritance and that inheritance of labyrinthic hardness of hearing is dominant. Atresia of the external auditory meatus, when bilateral, may result from a developmental anomaly related to hereditary occurrence. Acquired deaf mutism is generally the result of constitutional disorders, traumata, acute and chronic infections, and intoxications and otitis. Pathological anatomy may show malformation of the bone of the cochlea in association with defects of nerve tissue and malformation of the membranous connective tissue.

Beginning with Chapter VII, Lindenov presents his technique of examination, preparation of case record material and detailed comprehensive family and individual data. No clinical symptom was found that could be used as a means of differential diagnosis between hereditary and acquired deaf mutism. In the 32 families with deaf mutes including 58 hearing members who were studied, examination of the function of hearing and vestibular irritability showed that no clinical symptom was found that could reveal a trait for sporadic deaf mutism or on the whole distinguish hearing members of families with deaf mutism from other hearing persons. Statistics of the distribution of types of hereditary deaf mutism are given; 45.5 per cent of the total cases were of the hereditary type.

There is a significant relationship of deaf mutism and consanguinity of parents. Bottle feeding predisposes to such disorders as rachitis and otitis media which may produce deaf mutism. This was not true of hygiene and nutritional factors or alcoholism or tuberculosis in parents.

The material presented shows generally that sporadic deaf mutism is of monomeric, recessive transmission and that it is possible to determine deaf mutism of the heredolabyrinthic type. There seems to be evidence that there is a predisposition to acquire deaf mutism, but the details of its transmission are not entirely clear. There is no conclusive evidence of connection between either endogenous or exogenous hardness of hearing and deaf mutism. There is no correlation between deaf mutism and feeble-mindedness except in connection with retinitis pigmentosa. There were no cases of hereditary ataxia available for study. No relationship to other disorders was found. We quote Lindenov's statements on the prognosis of heredity of deaf mutism:

"Between two persons with sporadic deaf mutism, 100 per cent.

"Between a person with sporadic deaf mutism and one who is a carrier of a trait for sporadic deaf mutism, 50 per cent.

"Between two persons who are carriers of the trait for sporadic deaf mutism, 25 per cent.

"Between two persons with heredolabyrinthic deaf mutism, 75 or 100 per cent.

"Between a person with heredolabyrinthic deaf mutism and one without this disorder, 50 or 100 per cent."

Lindenov's study is an extremely significant contribution to the problem of hereditary deafness and the practical question of marriage and eugenics for the deaf. It merits wide and careful study not only by laboratory specialists but by those whose responsibility it is to guide the deaf. S. R. S.